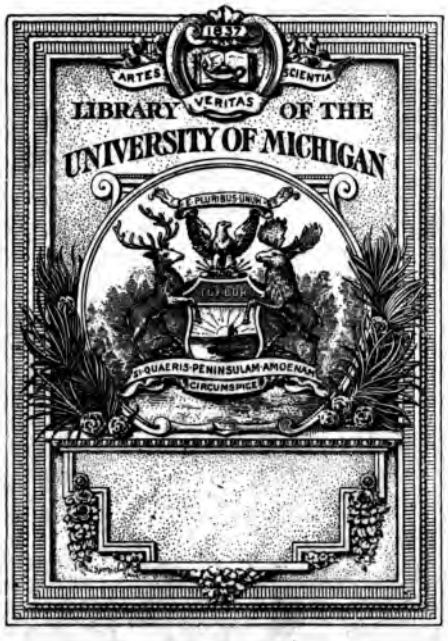
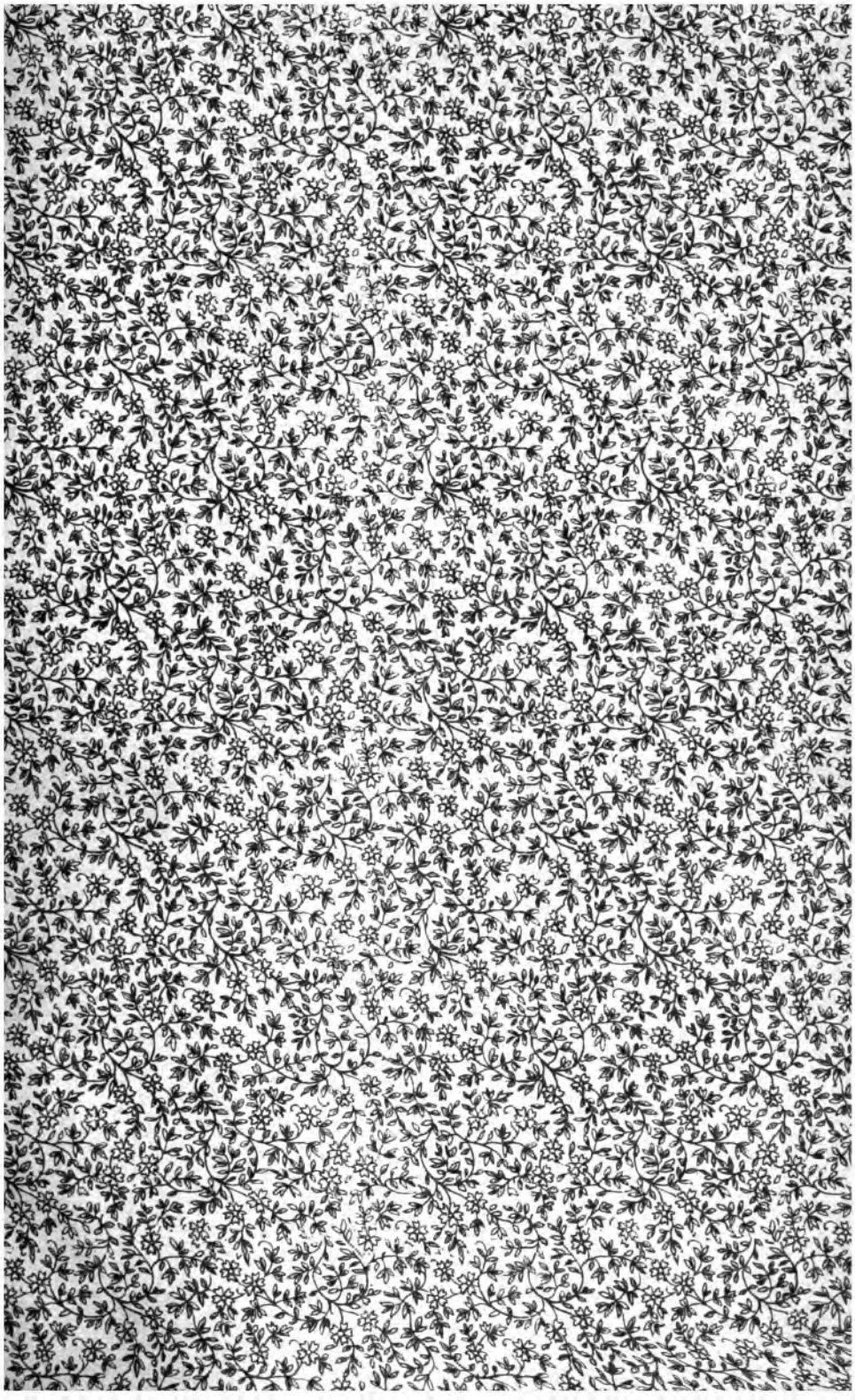

1837
ARTES
VERITAS
SCIENTIA
LIBRARY OF THE
UNIVERSITY OF MICHIGAN
E PLURIBUS UNUM
SI QUAERIS PENINSULAM AMOENAM
CIRCUMSPICE



PHILADELPHIA HOSPITAL

REPORTS.

VOLUME V.—1902.

EDITED BY

HERMAN B. ALLYN, M.D.

PHILADELPHIA:
PRINTED BY MAURICE H. POWER.
1903.

DEPARTMENT OF PUBLIC HEALTH AND CHARITIES,

HAVING THE CARE, MANAGEMENT, ADMINISTRATION AND SUPERVISION OF THE

CHARITIES, ALMSHOUSE, HOSPITAL,

AND ALL OTHER SIMILAR INSTITUTIONS, THE CONTROL OR GOVERNMENT OF WHICH IS INTRUSTED TO THE CITY.

DEPARTMENT OFFICE, 395 CITY HALL.

THE MAYOR OF PHILADELPHIA, HON. JOHN WEAVER.

DIRECTOR OF PUBLIC HEALTH AND CHARITIES, EDWARD MARTIN, M.D.

ASSISTANT DIRECTOR, MR. GEORGE W. SUNDERLAND.

PHILADELPHIA HOSPITAL.

SUPERINTENDENT, ROBERT H. SMITH.

CHIEF RESIDENT PHYSICIAN, M. H. BIGGS, M.D.

Medical Board of the Philadelphia Hospital.

ROLAND G. CURTIN, M.D., *President.* L. W. STEINBACH, M.D., *Secretary.*

PHYSICIANS.

ROLAND G. CURTIN, M.D.	S. SOLIS-COHEN, M.D.	ALFRED STENGEL, M.D.
J. H. MUSSER, M.D.	J. S. SALINGER, M.D.	HERMAN B. ALLYN, M.D.
F. P. HENRY, M.D.	THOMAS G. ASHTON, M.D.	DAVID RIESMAN, M.D.
W. E. HUGHES, M.D.	A. A. ESHNER, M.D.	T. MELLOR TYSON, M.D.

SURGEONS.

W. JOSEPH HEARN, M.D.	ORVILLE HORWITZ, M.D.	EDWARD MARTIN, M.D.
L. W. STEINBACH, M.D.	ERNEST LAPLACE, M.D.	J. CHALMERS DACOSTA, M.D.
ALFRED C. WOOD, M.D.	CHARLES H. FRAZIER, M.D.	

GYNECOLOGISTS AND OBSTETRICIANS.

BARTON C. HIRST, M.D.	J. M. FISHER, M.D.	W. FRANK HAEHNLEN, M.D.
EDWARD P. DAVIS, M.D.	R. C. NORRIS, M.D.	ELIZABETH L. PECK, M.D.
JOHN B. SHOBER, M.D.	GEORGE M. BOYD, M.D.	

NEUROLOGISTS.

CHARLES K. MILLS, M.D.	C. W. BURR, M.D.	WILLIAM G. SPILLER, M.D.
F. X. DERCUM, M.D.	F. SAVARY PEARCE, M.D.	CHARLES S. POTTS, M.D.

OPHTHALMOLOGISTS.

G. E. DE SCHWEINITZ, M.D.	CHARLES A. OLIVER, M.D.	HOWARD F. HANSELL, M.D.
	JOHN W. CROSKEY, M.D.	

DERMATOLOGISTS.

H. W. STELWAGON, M.D.	EMANUEL S. GANS, M.D.	MILTON B. HARTZELL, M.D.

LARYNGOLOGISTS.

GEO. M. MARSHALL, M.D.	CHAS. P. GRAYSON, M.D.	E. B. GLEASON, M.D.

ORTHOPEDIC SURGEONS.

H. AUGUSTUS WILSON, M.D.	J. P. MANN, M.D.	G. G. DAVIS, M.D.

PATHOLOGISTS.

W. M. L. COPLIN, M.D.	JOSEPH McFARLAND, M.D.	SIMON FLEXNER, M.D.

PEDIATRISTS.

WM. C. HOLLOPETER, M.D.	E. E. GRAHAM, M.D.	J. P. CROZER GRIFFITH, M.D.
	J. MADISON TAYLOR, M.D.	

BACTERIOLOGISTS.

L. NAPOLEON BOSTON, M.D. R. C. ROSENBERGER, M.D.

DENTAL SURGEONS.

R. H. NONES, D.D.S. M. H. CRYER, D.D.S. I. NORMAN BROMELL, D.D.S.
T. C. STELLWAGON, JR., D.D.S.

ANESTHETIZER.

CHARLES L. LEONARD, M.D.

ASSISTANTS TO MEMBERS OF THE STAFF.

JOSEPH SAILER, M.D. JOSEPH F. EVANS, M.D. J. W. McCONNELL, M.D.
J. NORMAN HENRY, M.D. J. B. CARNETT, M D. D. J. McCARTHY, M.D.
H. R. M. LANDIS, M.D. D. G. METHENY, M.D. T. H. WEISENBURG, M.D.
WM. PEPPER, JR., M.D. T. TURNER THOMAS, M.D. HORACE CARNCROSS, M.D.
J. W. CRUICE, M.D. MARY W. GRISCOM, M.D. HERBERT J. SMITH, M.D.
J. C. WOOD, JR., M.D. A. H. GRAHAM, M.D.

REGISTRARS.

B. F. STAHL, M.D. JOSEPH SAILER, M.D. W. A. N. DORLAND, M.D.
W. C. PICKETT, M.D. R. D. NEWTON, M.D. J. H. McKEE, M.D.
D. J. McCARTHY, M.D. T. H. WEISENBURG, M.D.

PREFACE.

The present volume is Volume V. in the series of Hospital Reports. While not as large as some of the earlier volumes, its prompt appearance should be such an encouragement to the contributors that the editor of future volumes will have a lighter task.

The wise policy of former editors has been followed, and historical articles bearing upon the development of the hospital or special departments of it will be found at the beginning of the volume.

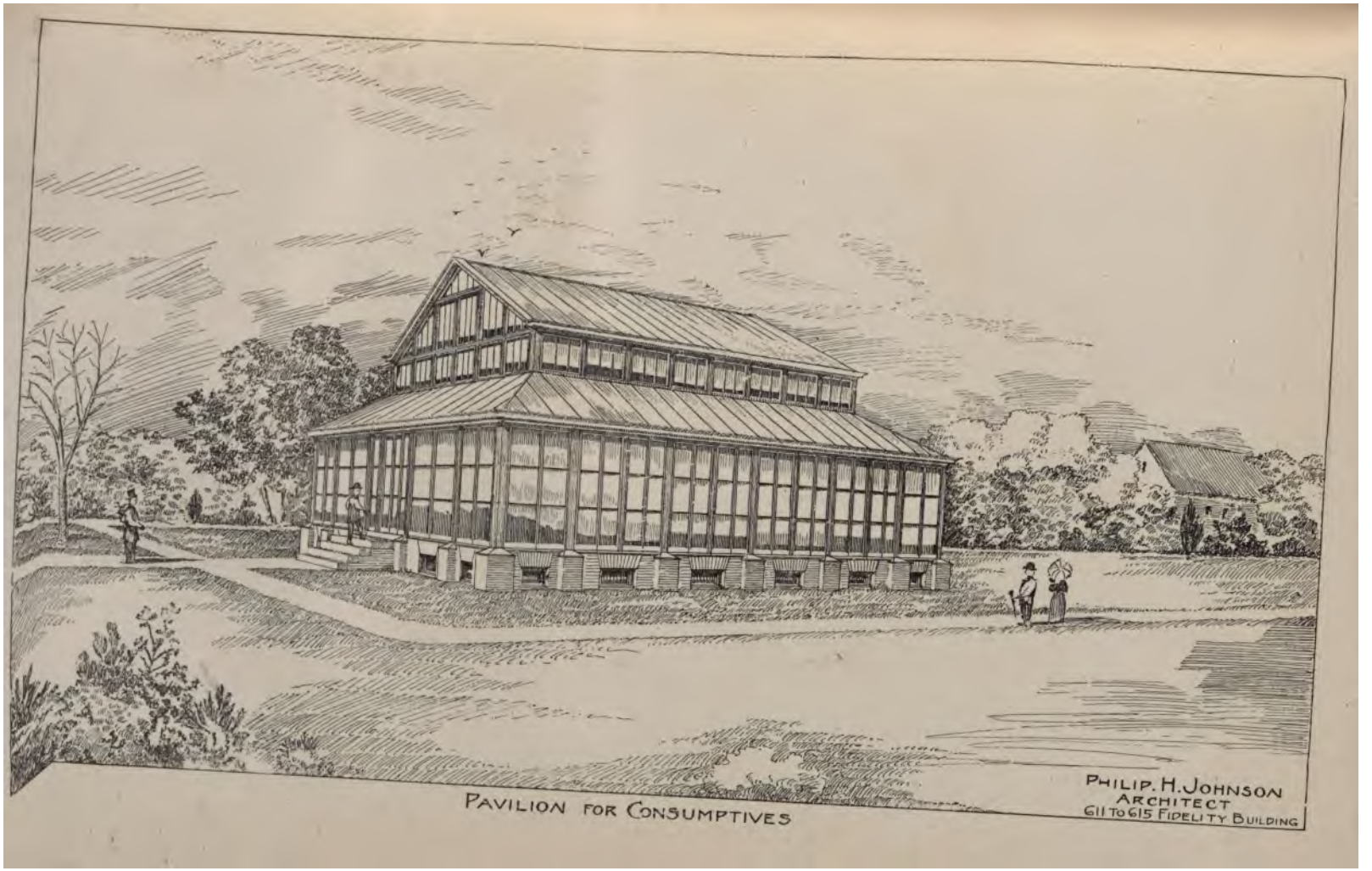
A glance at the table of contents shows that medical and neurological papers form about two-thirds of the whole number, doubtless because the clinical material in these wards is exceptionally rich. Probably nowhere in this country are the opportunities for the study of clinical medicine on its pathological side as good as in the medical wards at Blockley; and the nervous wards are unsurpassed for the study of neuropathology. From them alone the history of neurology in this city could be written.

We are fortunate in having in the present volume four papers from members of the ophthalmological staff. The contributions from the surgical, gynecological and obstetrical departments, and from the special departments, will become richer and more varied when once the much desired separation of the out-wards from the hospital is accomplished.

The editor wishes to thank the President of the Staff, Dr. Curtin, and Dr. Mills, for their encouragement and assistance, and the Publication Committee, Dr. de Schweinitz, Dr. Hughes, Dr. Frazier, Dr. Martin, Dr. Eshner, and Dr. Stengel, for their co-operation.

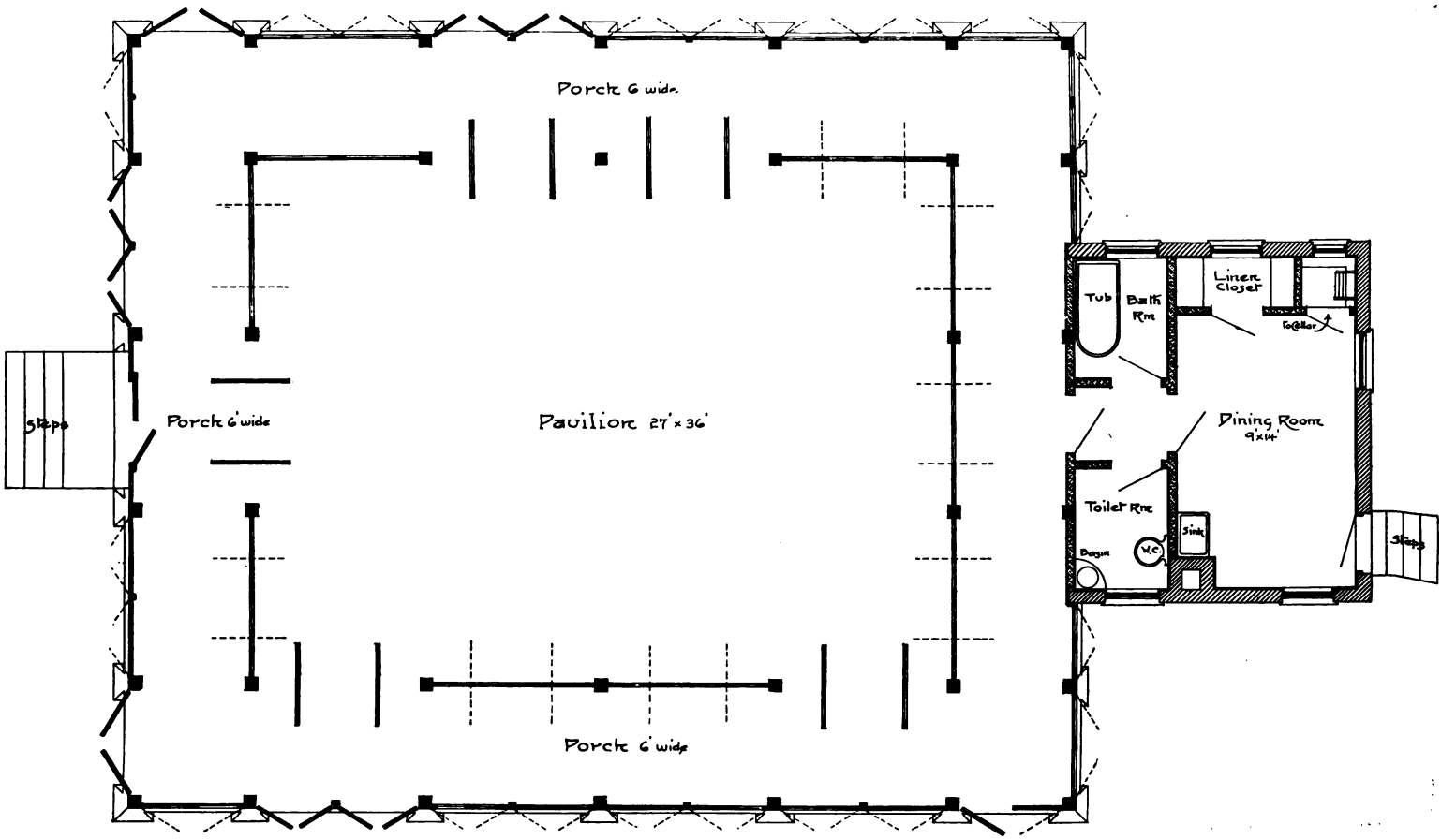
July, 1903.

CONTENTS.



PAVILION FOR CONSUMPTIVES

PHILIP. H. JOHNSON
ARCHITECT
611 TO 615 FIDELITY BUILDING


Porch 6 wide
Steps
Porch 6' wide
Pavilion 27' x 36'
Porch 6' wide
Tub
Bath Rm
Linen Closet
Cellar
Dining Room 9x14'
Toilet Rm
Sink
Basin
W.C.
Steps

PLAN

THE NEW PAVILIONS FOR CONSUMPTIVES AT THE PHILADELPHIA HOSPITAL.[1]

By JOHN V. SHOEMAKER, M.D., LL.D.,

PRESIDENT OF THE BUREAU OF CHARITIES AND CORRECTION; PROFESSOR OF THERAPEUTICS IN THE MEDICO-CHIRURGICAL COLLEGE, PHILADELPHIA, ETC.

The application of the most approved methods to the care of the poor consumptives is a subject which interests every large municipality and general hospital. It is, indeed, one of the questions of the day. Questions of finance, and limitations of space enter largely and necessarily into the problem. In this city the Philadelphia Hospital is the ultimate refuge of consumptives belonging to the poorest class. This institution has a long and admirable record, and has undoubtedly accomplished much good in the alleviation of disease. Its main buildings and general wards are, however, far from being well adapted to advanced methods in the treatment of tuberculosis. A modification of existing conditions has become necessary. The problem is to place our patients amidst surroundings capable of creating a strong impression upon the disease, and arresting its progress in cases not too far advanced. From the necessities of its position as a great city charity this hospital has long been overcrowded. Improvements are now being prosecuted which shall eventually separate the general hospital from the insane asylum and almshouse, and thus afford more ample space as well as far better facilities for the accommodation and treatment of the sick poor of Philadelphia.

There are always many consumptives in the wards of this institution. All periods are represented, but most of the patients are in an advanced stage of tuberculosis. It is not, as a rule, until they are unable to perform their accustomed work, and until all their resources have utterly failed, that they enter the hospital.

For the benefit of this unfortunate class a method which I have employed with marked benefit in private practice has been elaborated

[1] Part of a paper on "The Pavilion and Open-air Treatment of Tuberculosis," contributed to the Medical Bulletin, Philadelphia, January, 1903.

and adapted to usefulness on a larger scale. I have been in the habit, wherever circumstances permitted, of having my patients spend their days, during the milder seasons of the year, in good weather, upon balconies or verandahs built out from the house. In winter, or in stormy weather, hot-houses and conservatories were utilized when practicable. These structures are often arranged in such a manner that the glass sashes can be thrown widely open. According either to the state of the weather or the season of the year the patients could enjoy, for much of the time, the advantages of open-air life or the sun-parlor. From this germ, and after repeated consultations with the architect, Mr. Philip H. Johnson, of this city, plans have been perfected which will extend the advantages heretofore only within reach of the people in easy circumstances to indigent patients of the Philadelphia Hospital.

The intention is to erect pavilions which will permit the adoption of the open-air and sunlight treatment of tuberculosis. City Councils of Philadelphia have appropriated one hundred and fourteen thousand dollars, which will suffice for the construction of six sunlight pavilions, and contracts have been awarded for erecting them without delay.

The wood-work of hot-houses, conservatories, and verandahs constitutes a disadvantage from the fact that it becomes a lodging-place for pathogenic bacteria, and this capability would become all the more marked if numerous tuberculous patients, as in a large hospital, were continuously housed in such an apartment. But the pavilions we are contemplating are of unique design. Their original and distinctive feature is the material of which they are constructed. In brief, steel supports are substituted for wood. The structures will be composed entirely of glass, with the exception of steel frames to support the weight and a basis of cut stone and brickwork. The specifications call for materials of the best description. The co-operation of the architect and contractors provides for the stability and economical construction of the edifices; so that, as a whole, the work shall be a finished, complete, and unique one of its kind. The contractors are given to understand that the buildings must constitute a perfect piece of work, and the materials entering into it are to be first class in every respect. The contractor is required to brace properly and secure all parts of the work against wind, storm, and frost. All materials in the columns are to be of steel, all the rods and bolts of wrought iron.

The beams and girders are to be set at uniform distances between established centres. On all the columns brackets and connections are to be provided in order to secure first-class supports for the load. The steel used in the buildings is to be of the best make and uniform quality. All is to be tested in order to comply with the most stringent requirements. The walls and roofs are to be of plates of glass. Without entering any further into architectural details, the one great and distinctive object to be served by the columns, beams, connections, and other necessary structures is the support of glass sides which can be thrown open and a glass roof which can be raised. The glass sashes move on pivots placed in the supports. These act as air-tight joints; the plates of glass, when thrown open, lock tightly together and permit the entrance of a broad and free stream of air and an abundance of sunlight. Analogous arrangements are made for holding the roof firmly open. By these contrivances when weather and temperature are suitable the patients, even if bedridden, will obtain the inestimable advantages of an open-air treatment. In stormy weather and in winter they are protected from inclemencies. In fair, but cold, seasons the patients receive the benefits of a sun-parlor. The structures are perfectly air-tight, and draughts are excluded. An excellent system of ventilation has been adopted so that when the apartments are closed the air is changed and renewed every twelve minutes. Vitiated air is thrown out into the surrounding atmosphere. Due provision has been made for heating the rooms.

Each pavilion consists of one story, 28 feet in height. The dimensions of each are 48 x 39 feet. Between each two pavilions is an open space of 40 feet, and the six buildings are fronted by a space 600 feet long and 80 feet wide. The ground upon which the pavilions are placed is high and dry. It is to be planted with such shrubbery as will flourish in this location. The salubrious effects of foliage in purifying the atmosphere will be of service to our patients. Vegetation has also an important influence in equalizing the distribution of heat throughout the twenty-four hours.

The main room of each inclosed pavilion is the dormitory, which accommodates thirty patients. If necessary, the corridors can be included, and will give space for ten more patients in each pavilion. The floor is granolithic. Each pavilion contains a kitchen, lavatories, bath-rooms, linen-closet, and dining-room, 18 x 14 feet in extent.

sufficient to seat eighteen people at one time. Each bath-room is furnished with two tubs and two shower-baths. Patients can perform their own ablutions, or be bathed by attendants in the bath-rooms or sponged in their beds, according to their condition. The shower-baths are so arranged that the temperature and stream are regulated and varied simply by turning a handle.

Patients will be lodged in the pavilions in accordance with the stage of their disease, those of the same stage being placed together, as far as possible. When their sides and roof are thrown widely open the pavilions will be transformed, to all intents and purposes, into open-air gardens, in which the patients will inhale pure, fresh air and receive the salutary influences of the sun's rays. Those who are too ill to leave their beds will be relieved by the genial powers of air and sky. It is intended also to place in each pavilion a large, static electrical machine, in order that when they are inclosed ozone may be generated and add its renovating effect to that of the ordinary ventilation. This allotropic condition of oxygen is an efficient aid in promoting nutrition. It is also antidotal to the life and activity of disease-germs. Tubercle bacilli are less virulent in an atmosphere which contains a suitable proportion of ozone.

These are the broad general outlines of a project which will secure to the consumptives of the Philadelphia Hospital many of the advantages derived from change of climate which, in their cases, is impossible, and which will render these pavilions "hospital sanatoria," as they may truly be termed.

THE NEW BUILDINGS AT THE PHILADELPHIA HOSPITAL.

By HERMAN B. ALLYN, M.D.

Since the last REPORT OF THE PHILADELPHIA HOSPITAL was issued, four new buildings for hospital purposes have been erected, or are now in course of construction. These buildings are a children's hospital, a ward for venereal and skin diseases, pavilions for consumptives, and a new maternity ward. Thanks to the lively interest in the hospital taken by Mayor Ashbridge and City Councils, and to the energy and efficiency of Dr. Shoemaker and the Bureau of Charities, the prospects for the hospital were never brighter. Not only have these new buildings been completed or begun, but money has been appropriated and plans have been adopted for a new Philadelphia Hospital for the Insane, to be erected on the House of Correction grounds at Holmesburg, on the Delaware; and on the same grounds is to be erected a new Philadelphia Hospital for the Indigent. As soon as these new buildings are ready for occupancy, the present Insane Department and Almshouse will be removed to Holmesburg, and the West Philadelphia ground will be used entirely for hospital purposes, the hospital to be called The Philadelphia General Hospital. The writer feels that he is only voicing the feelings of the Medical Board when he expresses appreciation and grateful thanks to the city administration and the Bureau of Charities for the plans for the betterment of the hospital, plans which are so generous in conception and which have already been so successfully begun.

The Children's Hospital. This is a brick and stone building, situated across Pine street, opposite Thirty-fourth street, on the site of the former Children's Home, facing west. It is 46 feet high, and consists of three stories and an attic. There is a central or administration building, 35 x 46 feet, and two wings, 46 x 30 feet, which come off at right angles to the main building. The latter is entered at the street level, and on the first floor, on each side of the main corridor, are two reception-rooms, 14 x 12½ feet, a store-room, a large elevator, and further back, across a lateral corridor, a dining-room and kitchen. There is also an iron stairway to the second floor. On the

second floor, over the central portion, are two nurses' rooms and a reception-room and elevator. Beyond, over dining-room and kitchen, are a lavatory, a linen-room, and a large play-room, 16 x 16 feet. On each side of the central hall are wards, 36 x 30 feet, which open into sun-parlors, 8 x 30 feet, at the extreme north and south ends of the building. The third floor is a repetition of the second, except that on the north side is a group of eight private wards, 9 x 12 feet, with sun-parlor attached. In the central building, on the third floor, are a doctors' office with toilet-room attached, two nurses' rooms, an elevator, and a large operating-room, linen-room and lavatory. The operating-room and lavatory have granolithic floors, the rest of the floors are of wood.

Venereal Ward. This is a fire-proof building, consisting of two stories and a basement, of brick and stone construction and granolithic floors. It is 46 feet high. Externally, it has the appearance of one building, but a solid brick wall divides it in half, one portion being for male and the other for female patients. Communication from one part to the other is made by an iron passageway outside the building. The arrangement of rooms and wards is the same in each half of the building, so that it will be sufficient to describe one. In the basement are two large disinfecting-rooms and a linen-room. On the first floor are a lavatory, nurses' room with toilet-room attached, a dining-room for patients, an operating-room, doctors' office, and electric elevator. Adjoining these rooms, which are in the central portion of the building, are the wards for patients, 20 x 50 feet. The second floor is like the first, except that there is a small private ward accommodating from two to four patients. The large wards will accommodate twenty-four each. The granolithic floors will permit of thorough flushing and disinfection.

The Maternity House. This is a one-story building, 28 feet high, and of the same construction as the Venereal Ward. It is 79 feet long and 33 feet wide, and at one end is a sun-parlor, 33 feet long and 10 feet wide. There are a physicians' office with toilet-room attached, seven small rooms, averaging 12 x 12 feet, and an operating-room, 10 x 18½ feet. These rooms open on each side from a main corridor running through the building.

Dr. Shoemaker has already admirably described the new Pavilions for Consumptives.

HISTORY OF THE NERVOUS WARDS OF THE PHILADELPHIA HOSPITAL.

By T. H. WEISENBURG, M.D., AND D. J. McCARTHY, M.D.,
REGISTRARS OF THE NERVOUS DEPARTMENT.

The history of the neurological wards form an interesting chapter in the development of the study of neurology in this city and of this country. From the time of its foundation, in 1877, up to the present time, it has been the fountain from which has issued much of the literature on neurology supplied by Philadelphians. Possibly no hospital in the world has better facilities for the study of organic nervous disease and insanity than are to be found in this department.

In September, 1877, Dr. Charles K. Mills was appointed to organize and take charge of wards for nervous diseases. The out-wards were full of the richest material. We will here quote from Volume I. of the PHILADELPHIA HOSPITAL REPORTS: "The wards for nervous diseases were started in the old wooden pavilions in the clinic yard. The number of patients at first was limited, and these were collected by Dr. Mills from the out-wards, picking up here and there a case of hemiplegia, of sclerosis, or of some other form of organic nervous disease. Many such patients in a deplorable condition were found, some of them in the cubbies or blind-rooms. In the third story of the men's out-wards was also a room known as the 'paralytic ward,' in which were collected a number of helpless patients. This ward was included under the care of the neurologist, and after a long time its occupants were transferred to the pavilions. Gradually patients, both male and female, were added to the limited numbers first placed in the nervous wards, and as several of the pavilions were at that time unoccupied, no limit was put to the growth of the wards until, in 1890, the number of patients had reached two hundred and fifty or more."

In 1884 the women's nervous wards were removed to the lower floor of the women's out-wards. "In 1886 a new brick pavilion for the men's nervous wards was erected in the yard east of the south-eastern main buildings, parallel with it, and between it and the wooden

pavilions. In 1877 another one-story brick building was erected in the western side of the same yard, but at an angle with the first pavilion and the main building. This was the first of a series of pavilions which had been provided for by Councils and the Department of Charities and Correction. During the year 1888 the last of the rickety and unwholesome wooden pavilions were removed and two additional two-story brick buildings substituted for them. In 1889 an additional story was put on the pavilion erected in 1887." Since that time no additions or improvements have been made to these wards.

The growth of the wards is best shown in the following tabulated statements, showing the number of cases treated on the men's side and women's side during the year, and the number of patients remaining at the end of each year:

YEAR.	MALE PATIENTS.		FEMALE PATIENTS.	
	Treated.	Remaining.	Treated.	Remaining.
1877	No record.		11	11
1878	24	17	52	18
1879	28	12	98	26
1880	42	14	133	29
1881	56	14	156	35
1882	188	37	188	42
1883	No record.		220	37
1884	384	71	243	56
1885	397	124	204	62
1886	471	113	231	65
1887	525	120	280	61
1888	503	156	182	65
1889	462	162	172	66
1890	457	157	173	85
1891	520	192	189	78
1892	536	184	199	68
1893	544	232	195	78
1894	579	206	210	73
1895	547	191	217	79
1896	505	200	184	79
1897	454	206	189	85
1898	477	187	215	74
1899	479	185	182	82
1900	501	207	203	91
1901	658	237	257	89

The neurological department was reorganized so as to include the insane department in its joint jurisdiction in 1887. At the time of its reorganization the census at the close of the year 1887 showed 181

patients. The number has grown with considerable rapidity, so that at the close of the year 1901 it shows 326 patients for the nervous wards and 1487 patients for the insane department.

THE STAFF.

The neurological staff has been as follows: The first arrangement was that Dr. Mills should have charge of one-half of the whole number of patients admitted to the nervous wards, the care of the other half being distributed pro rata among the members of the medical staff on duty, these being four in number. In 1883 Dr. H. C. Wood was made an additional neurologist, and Drs. Mills and Wood remained in charge of the wards until 1887, when Dr. Roberts Bartholow was elected to succeed Dr. Wood. In 1887, the neurological department was reorganized upon a new basis, and its staff was now increased to four, Drs. Mills, Wood, Bartholow, and Francis X. Dercum. In January, 1888, Dr. Bartholow resigned, and Dr. James Hendrie Lloyd was chosen for the vacancy; later, in 1888, Dr. Wood also resigned, and Dr. Wharton Sinkler was elected to his place. When numerous changes were made in the medical board at the election in December, 1889, Drs. Mills, Dercum, and Sinkler were re-elected, and Dr. C. H. Bradfute was added to the staff in place of Dr. Lloyd. In the autumn of 1890 Dr. Bradfute resigned, and in December, Dr. Lloyd was re-appointed to his former position on the staff. When the reorganization of the nervous department took place in 1887 the entire insane department was included in its jurisdiction, the neurologists being also made visiting physicians to the insane department. This arrangement continued until 1890, when the neurologists, continuing in charge of the nervous wards, were made consulting instead of visiting physicians to the insane department, in which relation they have since remained.

Dr. Wharton Sinkler resigned in 1896, and Dr. Charles W. Burr was elected in his place. Dr. Lloyd resigned in the autumn of 1900, and the neurological staff was reorganized with the additions of Drs. F. Savery Pearce, William G. Spiller and Charles S. Potts. The first registrar to be appointed was Dr. Guy Hinsdale, who served from 1885 to 1892, when Dr. Augustus A. Eshner was appointed; he served until 1896, when he was transferred to the medical staff. Dr. B. F. Stahl succeeded him and served until 1900, when he was

appointed medical registrar. In 1900, Dr. W. C. Pickett was appointed and served until 1902, when he was made examiner for the insane, Dr. D. J. McCarthy being appointed in his place. On January 1, 1903, Dr. T. H. Weisenburg was appointed an additional registrar.

TEACHING.

From the time of their foundation the nervous wards have been used for teaching purposes by the different members of the neurological staff. Considering the abundant and excellent material on hand, no hospital in the country offers such splendid opportunities to the student. Dr. Mills first began to lecture on nervous diseases, in the usual Wednesday and Saturday clinics, in the year 1878. At first, when he was the only member of the neurological staff, he delivered as many as ten and twelve lectures, mostly in the spring. When other members of the staff were appointed the lectures were differently apportioned. During the administration of Dr. James W. White, when four members of the neurological staff were appointed, namely, Drs. Mills, Wood, Bartholow and Dercum, the number of lectures assigned to each man was about four to six. These lectures were distributed over the year, and they amounted to from sixteen to twenty-four in all.

In February and April, 1880, Dr. Mills gave a course of clinical lectures on insanity in the amphitheatre of the Philadelphia Hospital, drawing his material from the insane department of the hospital, through the courtesy of Dr. D. D. Richardson. These lectures on mental diseases were probably the first clinical lectures on insanity delivered in Philadelphia, with the exception that Dr. Isaac Ray,[1] "the greatest alienist America has yet produced, who honored Philadelphia by his residence for many years," had previously lectured on mental diseases at the Jefferson College, and Dr. D. D. Richardson, then Superintendent of the Insane Department of the Philadelphia Hospital, in illustration of Dr. Ray's lectures, had shown some cases of insanity at the hospital. It would be well to state in this connection an interesting fact, namely, that Dr. Isaac Ray was from 1870 to 1873 a member of the Board of Guardians of the Poor, which formerly had charge of the Philadelphia Hospital.

Dr. Mills gave ward instruction to students of the voluntary fourth

[1] Mills, C. K.: American Psychological Journal, vol. i., April 1, 1883.

year class of the University of Pennsylvania, which was established in the session of 1882–1883, also to students of the post-graduate course of the University, which lasted for only three years. He also, from time to time, after the foundation of the Philadelphia Polyclinic, gave ward and bedside instruction on nervous diseases to students of that institution, during the entire period of his connection with the college.

After Dr. Mills's election as Professor of Mental Diseases and of Medical Jurisprudence at the University of Pennsylvania, in 1893, in addition to clinical lectures on nervous diseases he also gave ward or bedside instruction on insanity and nervous diseases in the insane department and in the wards for nervous diseases.

In 1895–1896, as a part of his course on medical jurisprudence, he began to give clinical lectures on the medical jurisprudence of nervous diseases and insanity, these being probably the first clinical lectures in which an effort to teach medical jurisprudence was made. The lectures embraced neurological subjects of medicolegal interest, such as the traumatic neuroses, hysteria, hypnotism, aphasia, the effects of electricity, apoplexy, epilepsy, alcoholism, etc. The cases were first shown and clinically demonstrated, then the methods of examination for medicolegal purposes and the manner in which affections of this kind became of interest in medical jurisprudence were discussed. Cases illustrating the various forms of insanity were shown at these lectures, the methods of examining, especially for medicolegal purposes, detailed, and the modes in which the cases became of medicolegal interest were explained.

On the establishment of the fourth year course at the University of Pennsylvania, in the session of 1893 94, instruction in nervous and mental diseases was given by Dr. Mills, on cases drawn both from the nervous and insane departments, at the Tuesday and Thursday clinics.

Dr. Wood gave clinical lectures on neurology in the regular Wednesday and Saturday clinics of the hospital.

Dr. Dercum, from the fall of the year 1883 until the spring of 1892, gave ward class instruction in nervous diseases to students of the University of Pennsylvania. After his election as Professor of Nervous Diseases in the Jefferson Medical College he gave, from the fall of 1892 until the spring of 1896, ward class instruction to students of the Jefferson College, besides delivering clinics on neurology in the regular Wednesday and Saturday roster.

Dr. Lloyd instructed some special classes in nervous diseases besides the regular clinical teaching.

Dr. Sinkler also participated in the regular teaching of the hospital.

Dr. Burr has given regular clinical lectures, besides instructing students in the wards. Since his election as Professor of Mental Diseases in the University of Pennsylvania he has delivered his regular course on insanity in this hospital, drawing his material from the insane department.

Dr. Pearce gave a series of five special clinics upon nervous and mental diseases after his election to the neurological staff in 1900. He also gave ward instruction weekly, during his service, to students of the Medico-Chirurgical College, besides participating in the regular clinics. This has been repeated this session.

Dr. Spiller, during the session of 1901–1902, and also during the present year, lectured on nervous diseases to students of the Women's Medical College of Pennsylvania, and to students of the University of Pennsylvania.

Dr. Potts gave ward classes and clinics in the session of 1901–1902, and ward classes this present session.

Dr. Pickett gave demonstrations of insane cases to students of the Jefferson College every Saturday during the session of 1902–1903.

Dr. McCarthy lectured on clinical medical jurisprudence to students of the Women's Medical College and the University of Pennsylvania during the spring of the present year.

Literature.

Most of the important contributions to neurology that have been made in this city, and many, if not most, of the important neurological contributions made to medical literature in this country, have come from the nervous wards of the Philadelphia Hospital.

Dr. Mills, either alone or in collaboration with others, between 1878 and the present time, has published more than ninety articles in various journals, based upon the work done at the Philadelphia Hospital.

Among noteworthy observations made by Dr. Mills, as the result of cases studied in the hospital, are numerous observations on aphasia; one of the first cases of disseminated sclerosis, with microscopical examination, reported in this country; a considerable number of cases corroborating or fixing the location of centres of representation in the

cerebral cortex, as a case accurately localizing the auditory centre; cases showing unusual aberrations of fissural and gyral development; and cases with pathological investigations demonstrating new types of tract degeneration. In several cases brain tumors were first demonstrated by the Rœntgen rays in cases reported by Dr. Mills, in association with Dr. G. E. Pfahler, one of these being the second case on record. A large part of the clinical and pathological work incorporated in the volume of Dr. Mills's treatise on *The Nervous System and its Diseases*, dealing with diseases of the brain and cranial nerves, is based on investigations in the Philadelphia Hospital.

Most of Dr. Wood's observations in neurology depend upon material obtained in the Philadelphia Hospital. Dr. Wood was the first in this country to describe, from original observations, the clinical aspect of cerebral syphilis.

Dr. Dercum was the first to describe and point out the new disease, adiposis dolorosa, from a case in the nervous wards of the Philadelphia Hospital. He has published annually important contributions based on work done here. In the text-book on *Nervous Diseases by American Authors*, edited by Dr. Dercum, the Philadelphia members of the neurological staff contributing are Mills, Dercum, Lloyd, Sinkler and Burr. Much of the material and many of the illustrations were founded upon Blockley cases.

Dr. Lloyd contributed extensively to various scientific journals and text-books. His elaborate article in the *Twentieth Century Medicine* on the cerebrospinal and sympathetic nerves, constituting almost an entire volume, is based chiefly on Philadelphia Hospital work. Dr. Lloyd contributed an important work on localization, based upon the early work of the experimenters, Ferrier and Horsley. One of these cases was one of the earliest reported in America. It was a case of Jacksonian epilepsy which was operated on by Dr. Deaver, the cortex explored by electrical reaction and the epileptic centre excised. Besides, Dr. Lloyd reported important cases of syringomyelia, one of the first to be [illegible] traumatic lesions of the cervical region of the [illegible] were especially noteworthy as tending [illegible]rmal, and pain sense.

[illegible] of valuable articles. One of [illegible] because the two cases, on wh[illegible] first in this country in

which a diagnosis of syringomyelia was made and confirmed by autopsy.

Dr. Burr has made valuable annual contributions from material studied in this department. Among the most noteworthy of these are contributions to the study of stereognosis and its value in localization of cortical lesions; the pathology of adiposis dolorosa, and, in conjunction with Dr. McCarthy, the pathology and pathogenesis of acute internal hydrocephalus (meningitis-serosa).

Dr. Pearce, in the short time that he has been connected with this institution, has contributed some very valuable articles.

Dr. Spiller's work in the Pepper Laboratory of the University of Pennsylvania, on material furnished from these wards by Drs. Mills, Dercum, and Lloyd, antedated his official connection with the hospital. Among the most noteworthy of his many valuable contributions during that time, and since his appointment as visiting neurologist, the following may be mentioned: the localization of the motor area of the cortex, by studying the degeneration in a case of amyotrophic lateral sclerosis; the nervous manifestation of malaria of the central nervous system, reporting the first case of disseminated sclerosis with necropsy, due to malaria; a contribution to the localization of the sensory path in the internal capsule from a hemorrhagic lesion affecting the lenticular nucleus and the *Carrefour Sensitif*.

Dr. Potts has contributed extensively to the literature since his connection with the institution in 1901.

Dr. Pickett's statistical study of paresis from cases in the insane department in this hospital is a noteworthy contribution, among many others.

Dr. McCarthy, since 1899, has contributed annually from two to three articles based upon Philadelphia Hospital material; a recent one showing the relation of the ductless glands and the lympathic system (hæmo-lymph glands) to the disease adiposis dolorosa; also, with Dr. F. S. Pearce, a contribution to the anatomy of the median fillet, reported to the American Neurological Society, 1902.

Dr. Weisenburg has also contributed some articles.

Both the teaching and the investigations, which have resulted in widespread and incalculable benefit, not only to the patients of this institution, but to mankind in general, have been due to the hearty co-operation and support furnished at all times and in the most liberal spirit by the members of the Board of Charities and Correction.

A BRIEF SKETCH OF THE ESTABLISHMENT OF THE ORTHOPEDIC DEPARTMENT.

By H. AUGUSTUS WILSON, M.D.

In 1899 the surgical staff was composed of Drs. W. Joseph Hearn, L. W. Steinbach, Orville Horwitz, Ernest La Place, J. M. Barton, Edward Martin, J. Chalmers Da Costa, A. C. Wood, and Charles H. Frazier, among whom arose a desire for an orthopedic department, to which suitable cases could be referred and yet not deprive them of material for the clinics.

As a result of many conferences, a letter was drafted and sent to the Board of Charities and Correction, in which was stated an acceptible basis for the creation of this department. Experience has shown that the mutually agreeable arrangement has been carried out with satisfaction to all. For many years efforts had been made by others to have such a department, but always with the same negative result. When Dr. A. Sydney Roberts was a member of the surgical staff he arranged, through the courtesy of his colleagues, to confine his attention to orthopedic cases. The staff fully realized that the term of service of the surgeons was inadequate for the proper handling of cases which require observation for periods of many months, and often years. Repeated efforts at that time to have a separate department for orthopedic cases shared the same fate as all other attempts. The following extracts from the minutes of the Medical Board and from the minutes of the Board of Charities and Correction are fully explanatory:

OFFICE OF THE CHIEF RESIDENT PHYSICIAN,
PHILADELPHIA HOSPITAL,

January 8, 1900.

A meeting of the Medical Board of the Philadelphia Hospital was called for 5 o'clock this afternoon. Present were Drs. Curtin, McKelway, Horwitz, Hearn, Martin, Da Costa, Peck, Mills, Barton, Steinbach and Boston. Minutes of the meeting of October 9, 1899, were read and approved. The following communication from Dr. H. Augustus Wilson, directed to the Bureau of Charities and forwarded to this Board for consideration, was read:

Philadelphia, November 24, 1899.

BUREAU OF CHARITIES AND CORRECTION,

Gentlemen :—I desire herewith to apply for the appointment to the position of Orthopedic Surgeon to the Philadelphia Hospital, believing that such a position could be occupied to advantage for the patients without the slightest interference with the prerogatives of the members of the present staff. Permit me to suggest that, if I should receive the appointment as above, it will be perfectly satisfactory to me to receive for orthopedic treatment only such cases as were transferred to me by members of the staff on duty. Further, I believe that it would be conducive to continued harmony to have it clearly understood that I would be glad to have the surgeons on duty take any of my cases that they wished for their clinical lectures.

The orthopedic cases would probably be scattered through the men's, women's and children's wards, instead of having separate wards set aside for this class of cases. The modern orthopedic treatment of diseases and deformities of the bones and joints is occupying so important a field that operative surgeons do not, and cannot, give full attention thereto, and my experience leads me to believe that, under the arrangement herewith suggested, opposition would not only not occur, but cordial co-operation would be established. The expenses for establishing and maintaining this work could be kept well within bounds by abstaining from the use of expensive apparatus, relying upon plaster-of-Paris bandages, strips of iron and similar inexpensive and practical material. Trusting that this may receive your favorable consideration, I remain

Very truly yours,

(Signed.) H. AUGUSTUS WILSON,

1611 Spruce Street.

After the discussion upon the foregoing, upon motion of Dr. Martin, it was *Resolved*, That the appointment of Dr. Wilson as Orthopedist to the Philadelphia Hospital be recommended. The rights and privileges appertaining to the office being limited to those asked for and outlined in Dr. Wilson's letter.

(Signed.) L. W. STEINBACH, *Secretary.*

DIRECTORS' OFFICE,
DEPARTMENT OF CHARITIES AND CORRECTION,

Philadelphia, January 28, 1903.

DR. H. AUGUSTUS WILSON,

Dear Doctor :—Replying to your request of the 26th, would say that the following are extracts from our minutes :

December 29, 1899. *Resolved*, That the appointment of Dr. Wilson as Orthopedist to the Philadelphia Hospital be recommended. The rights and privileges appertaining to the office being limited to those asked for and outlined in Dr. Wilson's letter.

Same date, Dr. H. Augustus Wilson elected orthopedic surgeon.

February 13, 1900. *Resolved*, That the orthopedic surgeon shall have the care and treatment of such patients as may be referred to him by the members of the visiting staff.

Orthopedic cases under treatment by the orthopedist shall be cared for in their respective wards. Nothing in this rule shall be construed as preventing the members of the visiting staff, who refer the patient, from using the same for clinic purposes.

October 9, 1900. Teaching and ward instruction on diseases of the eye, nose, throat and ear, diseases of the skin, orthopedic surgery, and all specialties, will be arranged under the direction of the physician upon those subjects.

January 24, 1901. Orthopedic staff increased to three members. Dr. J. P. Mann and Dr. G. G. Davis elected.

Very truly yours,
JOHN V. SHOEMAKER, *President.*

RULES GOVERNING THE BUREAU OF CHARITIES, 1902.

Rule LXIII, Section 3. "Orthopedic surgeons shall have charge of all diseases and deformities of the bones and joints."

This rule when viewed by itself would appear to abrogate the conditions upon which the orthopedic department was founded, as it appears to give to this department a very large scope, but reference to the rules for the other departments will show that misunderstandings and conflict can only be avoided by a strict adherence to the letter and the spirit of the conditions upon which the department was originally established.

The rules state, "Surgeons shall have charge of all surgical cases. Gynecologists shall have charge of all diseases of the pelvic region. Pediatrists shall have charge of all diseases of children, from the time the mother passes from her puerperal state until they reach the age of puberty."

The scope of orthopedic surgery has never been absolutely defined, nor can it be, for the reason that it necessarily includes many conditions of deformities that had their origin in diseases that very properly came under the observation of other departments. For instance, rhachitics, tubercular disease of the pelvic bones, contractures of tendons in paralytic patients, rheumatoidal and gonorrhœal arthritis, fractures and dislocations resulting in deformities, etc., etc.

The generally accepted definition of orthopedic surgery is, that it is that branch of the practice of medicine which has for its object the prevention and the correction of bodily deformities, gives emphasis to the prevention of the deformity as well as to the prevention of the progress of same as being of equal or greater importance than the correction.

The continued cordial relations between the members of the orthopedic staff and their colleagues of other departments is abundant evidence of the wisdom of having established methods that avoid confusion and constantly tend towards harmony, with the object of the best good of the patient.

HISTORY OF THE ORGANIZATION OF THE ORAL SURGICAL STAFF OF THE PHILADELPHIA HOSPITAL.

By M. H. CRYER, M.D., D.D.S.

Dr. John V. Shoemaker, Surgeon-General of the State of Pennsylvania, had noticed at various times, especially during the war with Spain, the necessity of having dental services for the soldiers both in camp and in the hospital. He was compelled to ask for such voluntary assistance as could be obtained, there being no provision made by the State for such services, which, according to the writer's opinion, should be corrected by the State Legislature. Through Dr. Shoemaker having this experience, and his position as President of the Board of Charities and Correction, he examined into the condition of the patients under the care of the said board, and found that they required dental services which could not be satisfactorily rendered without a fully-equipped dental staff and internes. It was for this reason that he suggested and recommended to the Board of Charities and Correction the establishment of the staff, etc., and the writer wishes to state that Dr. Shoemaker has assisted the staff in every possible way.

The Oral Surgical Staff of the Philadelphia Hospital was organized in January, 1901. Its members having been appointed by the Board of Charities and Correction in December, 1900, and who still hold their positions, consist of Dr. Robert H. Nones, Dean and Professor of Mechanical Dentistry of the Medico-Chirurgical College; Dr. M. H. Cryer, Professor of Oral Surgery of the Department of Dentistry of the University of Pennsylvania; Dr. I. N. Broomell, Professor of Dental Anatomy and Prosthetic Technics of the Pennsylvania College of Dental Surgery, and Dr. Thomas C. Stellwagen, Jr., Lecturer on Physiology and Physical Diagnosis in the Philadelphia Dental College.

The first oral surgical interne was Dr. W. David Easton, who was appointed at the time of the organization and served until June, 1902, at which time Dr. Robert H. Ivy was appointed and is still on duty. *Since his appo*intment two internes have been on duty with the excep-

tion of a short time. The work accomplished by these internes, as the full report will show, has been considerable. As the department develops, more internes will doubtless be found of great advantage. One of the present internes, in company with Dr. W. W. Hawke, physician in the department for insane, made an examination of the patients' mouths, and found nearly three hundred who require immediate dental services.

Doctors of dental surgery are not so easily obtained for internes as doctors of medicine. Although a large number of dentists are graduated every year in the city of Philadelphia, there is such a demand for good dentists as assistants, not only in the United States but in foreign countries, that most graduates begin practicing at once. This, of course, makes it difficult to obtain dental internes. But I trust that as the facilities of this department increase and it becomes better known, there will be an increase in the number of applicants. In fact, I find there is already an increase in the inquiries for this position, usually from the best students in the colleges.

The duties of the staff and internes are to take care of the teeth and maxillary bones of the patients of the hospital, also to give public clinics for the students attending the various colleges in Philadelphia.

After the organization of the oral surgical staff, an appropriation was made by the city government to cover the expenses incurred in fitting up an operating-room in the hospital with all modern appliances for the treatment of teeth and diseases of the maxillary bones. In addition, a mechanical laboratory was constructed and supplied with all necessary equipment used in prosthetic dentistry and in making splints for fractured bones and also for the construction for cleft-palate.

The first clinic was given in the amphitheatre of the hospital March 9, 1901, with a good attendance. Since then forty-three clinics have been given with an average attendance of 93. For further particulars see report following. Ten clinics have been given since October 4, 1902, with an average attendance of $171\frac{7}{10}$. The total number of patients presenting for dental or oral surgical services has amounted to 3146, as shown in the detailed report.

In this session, 1902–1903—the dental college term runs from October to the following spring or summer—the names of the attending students are being recorded. One of the most interesting facts which has developed is the cosmopolitan character of the students in attendance.

Besides names from nearly every State in the Union, there are students from Japan, Netherlands, Ecuador, Australia, Canada, Switzerland, Sweden, Brazil, Germany, Nicaragua, New Zealand, France, China, Russia, England, Mexico, Chile, Cuba, Spain, Jamaica, Austria, Porto Rico, and Italy.

The first and principal object of the dental staff is to relieve the suffering caused by diseases of the teeth and maxillary bones. The second is to encourage scientific investigation of these diseases and to give opportunity for study such as cannot be obtained in the dental schools of this city. This will be best accomplished by means of laboratories equipped with proper appliances for carrying on original research. As soon as the hospital is separated from the almshouse, we hope that we may be provided with a building or suite of rooms which could be devoted exclusively to such laboratories.

When it is understood that a large number of graduates of foreign dental schools come to Philadelphia to take post-graduate courses, in addition to those who come for the regular course, the importance of our city as a centre of dental knowledge becomes apparent. At this time there are over three hundred foreign dental students here. It is our endeavor to give them all the advantages of clinics and laboratories possible. As many of our medical graduates go to Vienna, Paris, London, etc., for their post-graduate work, so we hope to attract the foreign graduates of dentistry by our more complete equipment and more thorough investigation in oral anatomy and surgery; by greater excellence in practical demonstrations upon patients of the proper treatment and filling of teeth, the making of artificial dentures and treatment of the maxillary bones, the substitution of lost tissue as in cleft-palate or other portions of either jaw, and of every operation which comes under the definition of oral surgery—the treatment by surgery or medication of lesions within the mouth or associated parts which may arise from dental diseases, and the treatment of an associated part which may cause a diseased condition of the mouth or jaws.

At the last meeting of the Pennsylvania State Dental Society, the president made the following statement: "The city of Philadelphia has recognized the dental profession by having four dental or oral surgeons placed on the staff of the Medical Board of the Philadelphia Hospital. At the city's expense dental laboratories and operating-

rooms have been fitted up in the hospital, which has two resident dentists. The dental staff and residents are received on equal terms with all others connected with the hospital." Then followed a report for the year ending May 1, 1902, giving an idea of what is being done in the Dental Department of the Philadelphia Hospital.

The committee, to which was referred the President's Address, made the following report in regard to the Philadelphia Hospital:

"We commend the action of the Board of Charities and Correction of Philadelphia in establishing a department of dental service in connection with the Philadelphia Hospital, upon terms of professional equality with other branches of medical and surgical services in that institution.

"We recognize in this action a practical appreciation of the value of efficient dental service in its bearing upon the public health, a fact which is fully borne out by the excellent exhibit of results set forth in the annual report of the resident dental surgeon submitted in the address of the president of this society. We take pride in the fact that Philadelphia has taken the initiative in this matter, and we trust that her example may be generally followed by all municipalities in their efforts to alleviate the physical ills of the indigent and dependent portion of their population."

The International Dental Federation, which has its headquarters in Europe, sent out blank forms last May to various dental organizations throughout the world, asking for information in regard to arrangements made by city governments for the care of the teeth of the poor.

It was a pleasure to be able to give the information as to what the city government of Philadelphia was doing for this class of patients in the Philadelphia Hospital.

In October, 1902, Dr. Florestan Aguilar, dentist to the King of Spain, came to America, instructed by his government to inspect the various dental colleges throughout the United States with the object of bettering the dental schools of Madrid. It was a pleasure to have him visit the Philadelphia Hospital and see the room and laboratories in which our dental work was done for the inmates of the hospital. I told him of the oral surgical clinics given by the dental staff to the students of the various dental schools of this city, and he expressed his hearty approval of the idea.

Dr. Aguilar spent two years in this city, from 1889 to 1891, study-

ing dentistry, and received his degree of Doctor of Dental Surgery from one of our city colleges.

Department of Oral Surgery, Philadelphia Hospital.

The following is a summary of the work done in the Department of Oral Surgery, Philadelphia Hospital, from March 1, 1901, to December 20, 1902:

Total number of patients presenting themselves for dental or oral surgical services from May 1, 1901, to December 20, 1902	3146
Roots extracted	1502
Teeth extracted	1235
Total number of extractions	2737
Fillings, temporary and permanent	482
Full dentures	60
Partial dentures	27
Inter-dental splints	11
Partial splint made for maxilla covering six anterior teeth	1
Porcelain inlays	50
Administrations of nitrous oxide	180
Administrations of nitrous oxide previous to the general anæsthetic ether	4
Administrations ethyl chloride	31
" ether	17
" cocaine	11
Impressions of forearm taken and casts made for same	3
Cases of simple fracture mandible successfully reduced	8
" compound fracture mandible " "	2
" double fracture mandible " "	1
" compound fracture mandible discharged without result, as patient refused treatment	1
" simple fracture superior maxilla	1
" dislocation mandible, left side successfully reduced	1
" chronic neuralgia cured by removal of infra-orbital nerve	1
" ankylosis, false, successfully treated	4
" alveolar abscesses	90
" abrasion treated	20
" canker sores	3
" difficult eruption of third molar successfully treated	6
" devitalized pulps " "	37
" epulis " "	2
" erosion " "	4
" fistula " "	11
" facial neuralgia " "	4
" marginal ginivitis " "	52
" specific ginivitis " "	66
" glossitis " "	2
" hypercementitis " "	1

Cases of	necrosis maxillary bone	successfully treated	11
"	pericementitis, acute	" "	17
"	pericementitis, chronic	" "	2
"	pericementitis, traumatic	" "	9
"	pulpitis	" "	47
"	pulp exposures	" "	33
"	putrescent pulps	" "	16
"	lead-poisoning	" "	1
"	diseased antrum	" "	3
"	cleft-palate abturator made		1
"	crown reset		1
"	external fistula successfully treated		1
"	hemorrhage " stopped		1
"	pulp canals " treated		58
"	pyorrhœa alveolaris treated		86
"	perforated roots successfully treated		9
"	scorbutus " "		1
"	salivary calculus " "		306
"	sensitive dentine " "		22
"	stomatitis, ulcerative " "		27
"	stomatitis, mercurial " "		69
"	stomatitis, smokers' " "		19
"	sebaceous cyst " "		1
"	epithelioma removed		1
"	fibro-lymphoma successfully treated		1
"	ankylosis " "		2
"	fracture of alveoli treated		3
"	hypertrophied gum successfully treated		4
"	plates repaired		10

There have been forty-one public clinics held by the dental chiefs, with an average attendance of 93 students, given as follows:

			ATTENDANCE.
March	9, 1901	Dr. M. H. Cryer	
"	16, "	" "	
"	23, "	" "	
"	30, "	" "	
April	8, "	Dr. Thomas C. Stellwagen, Jr.	
"	13, "	Dr. M. H. Cryer	
"	20, "	" "	
"	27, "	Dr. Thomas C. Stellwagen, Jr.	
May	4, "	" " "	
October	5, "	Dr. M. H. Cryer	
"	12, "	" "	
"	19, "	" "	
"	26, "	" "	
November	2, "	" "	
"	9, "	" "	
"	16, "	" "	

			Attendance.
November	23, 1901	Dr. M. H. Cryer	
"	30, "	No clinic	
December	7, "	Dr. R. H. Nones	
"	14, "	" "	
"	21, "	" "	
January	11, 1902.	" "	
"	18, "	" "	
"	25, "	" "	
February	1, "	Dr. M. H. Cryer	
"	8, "	" "	
"	15, "	" "	
March	1, "	" "	
"	8, "	" "	
"	15, "	" "	
"	22, "	" "	
October	4, "	" "	112
"	18, "	" "	100
"	25, "	" "	168
November	1, "	" "	213
"	8, "	" "	177
"	15, "	" "	143
"	22, "	" "	228
"	29, "	No clinic (Thanksgiving).	
December	6, "	Dr. R. H. Nones	227
"	13, "	" "	147
		Total	1515

The attendance at the last ten clinics averaged $171\frac{7}{10}$.

The following remarks were made by Dr. Easton, the first oral interne, at the conclusion of his report for May 1, 1902:

Red Stain. It has been suggested by someone that the peculiar red stain which appears upon the teeth of some people indicates predisposition to insanity, and that it denotes hereditary tendencies of that character. I have been interested to observe the mouths and to gather data in cases that have come under my care, and have made an examination of over 500 inmates of the department for the insane with the view of substantiating the supposition or disproving it.

In all I have found 71 cases of marked red stain; of these, 54 were male, 17 female, and of the whole number only 8 were insane. Most of the patients were neurasthenics. Of the 63 hospital cases, none gave any family history of insanity. Attempts to grow cultures in any medium failed. Microscopic preparations present a picture similar in character to that of the green stain, and the enamel is softened under

it. It is recurrent. Thoroughly removing it from the teeth does not stop the stain from reappearing.

Decayed Molars in the Insane. In a recent number of the *Dental Brief* there appeared a short paragraph in which someone asserted that badly decayed teeth, particularly the molars, were the cause of more people becoming insane than anything else. This is not so. I have, since reading the above, taken particular interest in examining the teeth of the insane and comparing the conditions found with those of inmates of the hospital side of this institution. While the mouths are equally unclean, I think that, taken as a whole, the inmates of the insane department have better teeth than have the hospital patients. In the matter of suffering pain insane people are analgesic.

Pyorrhœa Alveolaris. I have found fewer cases of pyorrhœa alveolaris among the inmates of this institution than I expected. Such cases as have come under my observation were of long standing, and every manner of treatment before extraction has been tried, but without result other than making the patients' breath less offensive.

Mercurial Stomatitis. Excellent opportunity for studying this condition is found in the venereal wards of the hospital. Briefly stated, my observations in regard to this form of malady are as follows:

That in almost every instance where patients are sent to me to have the mouth examined and cared for before specific treatment is commenced the stomatitis has not appeared.

That in many cases where the malady has already developed it readily responds to proper treatment.

The method of procedure in all instances is to thoroughly clean the teeth, remove all decaying roots, fill all cavities, and see that there are no rough edges to irritate the tongue, and that the patient keeps the mouth and teeth clean. Order mouth-wash, astringent. I have found that a 5 per cent. solution of potassium permanganate gives the best results in cases where the malady has developed.

Respectfully submitted,

W. DAVID EASTON,

Resident Dental Surgeon.

DR. DANIEL E. HUGHES.

1850–1902.

Dr. Hughes was born at Williamsport, Pennsylvania, in 1850, and was graduated from Jefferson Medical College in 1878. He became Chief Clinical Assistant in the medical out-patient department of Jefferson Medical College Hospital, but later he returned to his native city, and was appointed Chief Surgeon to the Philadelphia and Erie Railroad Company. In 1890 he was elected Chief Resident Physician to the Philadelphia Hospital, and it was while serving in this capacity that he was stricken with his final illness, death from tuberculous peritonitis taking place on October 28, 1902. He was the author of a Compend of the Practice of Medicine, which passed through numerous editions.

Dr. Hughes had a warm heart, a cordial manner, a large and handsome physique, and an attractive personality. For fourteen years he gave his best services to the hospital, filling a difficult position to the satisfaction of the Department of Charities, and winning the respect of the Staff and the affectionate regard of the patients.

A tablet in bronze, commemorative of the worth and services of Dr. Hughes, will be given suitable place at some future time.

CATARRHAL PANCREATITIS AND A STUDY OF ITS PATHOLOGY.[1]

By ROLAND G. CURTIN, M.D.

As far as I can learn from the literature on the subject at my command, the following comprise the recognized inflammatory diseases of the pancreas: Acute primary pancreatitis (sometimes suppurative), chronic interstitial pancreatitis, and parenchymatous pancreatitis. The pancreas is sometimes the seat of secondary inflammation, owing to inflammation of the surrounding organs. Writers on this subject inform us that this organ may be indurated, softened, hypertrophied, or atrophied; that it may undergo calcareous, fatty, tuberculous, cancerous, and fibrous degeneration.

It is, perhaps, unnecessary to state that the function of the pancreas is to furnish pancreatic juice, which is largely used to emulsify the fatty food, preparing it for absorption by the lacteals. After absorption, it goes to the thoracic duct, through which it passes to the left subclavian vein, where it enters the venous circulation. In any disease interfering greatly with the proper secretion of the pancreatic juice, digestion of the fatty food is imperfect; and, as the result, oil or fat is found floating on the stools. Hence, fatty diarrhœa is said to be diagnostic of disease of the pancreas.

The history of a case here given suggested this paper:

P. C., a native of Ireland; by occupation, a gardener. No history of tuberculosis, syphilis, or intemperance. He had lived for years in a malarious district, and many years before his death had shown symptoms of poisoning from that cause. During the fourteen years immediately preceding his death, he had not been entirely well. About this time he began to experience occasional severe, colicky attacks of pain in the abdomen; and between these attacks, sometimes a dull pain and a sense of weight and uneasiness in the epigastrium. Four years before his death he had acute articular rheumatism, followed by shortuess of breath after any exertion. Three years ago swelling of the feet was noticed, and was attributed to articular rheumatism.

About the same time, diarrhœal symptoms, with anemia, presented themselves.

[1] Read before the Pennsylvania State Medical Society, at Lancaster, in 1881, but never published. A description of a hitherto undescribed disease.

These increased progressively until a year before his demise, at which time he was obliged to take to stool a few moments after eating, the desire coming on so suddenly that his drawers sometimes suffered from accidents while he was fleeing to the water-closet. He frequently had as many as thirty stools in twenty-four hours. Neither blood nor pus had ever been noticed in his passages. About two months before his death his wife observed some hardened fat floating on a stool that had been passed some time before.

When I was called in his condition was as follows: His mind was fatuous, and debility was so marked that he was unable to rise either without swooning or without a tendency to it. The whole body was puffed from œdema, which masked any emaciation that might have been present. His cheeks and eyelids were puffy from the dropsical effusion, and there was decided pitting on pressure over the tibiæ. He was very anæmic, the tissue being white and translucent, like wax or alabaster.

The area of the impulse of his heart was increased, as was also the force. Over the mitral area there was a marked and harsh systolic murmur, transmitted to the left axilla, and heard posteriorly at the lower angle of the left scapula. Over the aortic valves, and along the aorta and the large blood-vessels, was heard a soft systolic murmur. There seemed to be very little evidence of disturbance of the circulation. The patient complained of dull pain and a sense of weight at his epigastrium, extending to the left hypochondrium. His diarrhœa was the most remarkable that I have ever seen.

A tablespoonful of a mixture of equal parts of limewater and milk would seemingly be evacuated per rectum in ten or twenty minutes, or even in less time, although he was resting quietly on his back in bed. The same was true of small quantities of food, drink, or medicine. Opium, nitrate of silver, bismuth, astringents, or any of the medicines usually given in cases of diarrhœa, were evacuated in the same speedy manner, and thus failed to give any relief. They seemed to be hurried through so rapidly as not to have sufficient time to be absorbed. It was "touch and go" with everything that went into his stomach or bowels. His urine was scanty, but was found to be free from albumin or sugar. He constantly grew weaker until the hour of his death.

Postmortem Findings.—The pancreas was enlarged to twice its normal size at the two extremities; the intermediate portion was very little more than its natural size. The whole organ was indurated, more especially at the two enlarged ends.

The left ventricle of the heart was somewhat dilated and hypertrophied. The mitral valve was thickened and bound down by the results of old endocarditis, but otherwise the heart was apparently healthy. The spleen, also, was enlarged to twice its usual size; while the stomach, intestines, liver, and lungs, were normal.

The tissues were remarkably free from adipose matter. The thoracic duct was not pressed upon by any organ. All the organs were healthy, except as above stated. Extreme pallor in all the tissues, as well as a slightly edematous condition, was noticed.

I saved the head of the pancreas for microscopic examination, and handed it to my friend, Dr. E. O. Shakespeare, at that time Pathologist of the Philadelphia Hospital, who kindly furnished the following report:

"The specimen, consisting of the head of a pancreas, that was placed in my hands for examination, was found to be considerably indurated.

"It was immersed in dilute alcohol for a few days. Subsequently a section was

made through the piece. This was stained with carmine and placed under the microscope for observation. The cut was seen to include a number of acini of gland lobulæ and sections of the smaller ducts leading to them. These small ducts presented the following appearance:

"The lumina of many were quite occluded by an accumulation of cellular elements, which were in the main, the offspring of the epithelial lining of these tubes. In other cases, the calibre of the ducts was greatly narrowed by proliferation of their epithelial lining. In a word, there was an evident and quite marked catarrh of the ducts. The basement membrane of these ducts was somewhat thickened; and here and there the endothelial cells separating it from the deepest layer of the lining epithelium were distinct, and evidently in a state of irritation. Behind the points of occlusion of the ducts, already described, the ducts and some or all of their branches often presented, in places, a slight, but decided dilatation, approaching the formation of a microscopic cyst.

"In the acini of the lobules of the gland, some traces of slow inflammation—or, at least, irritation—were visible. The gland cells occasionally contained two or more nuclei. Sometimes a kidney-shaped nucleus with a nucleolus in each end could be well seen. The protoplasm of the cells was usually slightly cloudy, but rarely sufficiently granular to quite obscure the nucleus. These cells were certainly neither in a state of fatty degeneration nor of marked atrophy. The trabeculae of the connective tissue separating the acini of a lobule were slightly thickened, and in a state of commencing cellular hyperplasia. The bands of interlobular connective tissue were in a very similar condition. Judging from the apparent age of these various lesions observed in the section from the head of the pancreas, it seems probable that the catarrh of the minute ducts was the primary trouble."

The history just given presents the following prominent symptoms: General dropsy, with organic cardiac disease; stomachic and intestinal dyspepsia, as shown by gastric symptoms; and diarrhœa, with occasional fatty stools. These symptoms were associated with extreme debility. The dropsy was general; not, for the most part, seeking the most dependent tissues, like the legs when standing, and the lower part of the lungs when in a sitting posture. The patient's legs were not much more edematous than his face, and the lungs were not particularly affected. To what was this dropsy due? The kidneys were not diseased, so that kidney dropsy is to be excluded. Pressue by the enlarged pancreas upon the ascending cava would not cause general edema. The condition of tne heart was not such as to forecast dropsy, since the circulation of the lungs, veins, and capillaries was not much interfered with. The heart trouble was mitral regurgitant disease and dilatation of the left ventricle, with an amount of hypertrophy that compensated for the weakness of the valves and of the left ventricle. The anæmic state of the patient and his starved condition were sufficient to account for the dropsical symptoms.

The stomachic and intestinal dyspepsia, we shall next consider. The diarrhœa appeared before the dyspeptic symptoms, associated with colicky pains in the abdomen. The heart disease was not the cause of the dyspepsia or of the diarrhœa, for the rheumatic attack that apparently caused the valvular disease, which was undoubtedly the first lesion of the heart, occurred only four years before the death of the patient. The diarrhœa began about fourteen years previously to his death. At first he had occasional slight attacks, but these grew worse and worse until he died. I have never seen a case of diarrhœa in which the stomach and intestines

were so irritable as in the latter stages of this one. As stated in the history, a tablespoonful of a mixture of equal parts of milk and limewater; a tablespoonful of water with a powder of pepsin and bismuth; a like quantity of milk-punch with limewater; a pill of opium or nitrate of silver and opium, washed down with a little water, or even a little port wine, would cause an evacuation in ten or twenty minutes. None of the foregoing remedies seemed to have the slightest effect; the same was true of vegetable or mineral astringents, and of any of the medicines usually given for diarrhœa. In the latter stages of his disease, the stools were sometimes noticed to contain fatty or oily particles of matter. Such a condition of affairs could not fail to result in death from slow starvation. In a case of simple pancreatic obstruction with good stomachic digestion, the fat is to a great extent isolated from the food digested in the stomach. The explanation of the fact that so little fat was found in the stools of our patient is, I think, that the stomach had failed to isolate the fat from the other food. We must also recollect that the secretion of the pancreas had not entirely ceased.

The usual effect of chronic diarrhœa is ultimately to produce stomachic disorder. This is due to two causes: first, the impoverishment of the blood. Poor blood cannot produce good gastric juice. Deteriorated gastric juice ultimately causes dyspepsia. Second, diarrhœal irritation or inflammation. This is prone to travel upward, increasing the peristaltic movements of duodenum and the stomach, thus hurrying the food through the upper part of the alimentary canal. These two causes, acting in concert, would sufficiently account for the subsequent dyspepsia of the patient.

The extreme debility was not due to the heart disease, for we have already demonstrated that to be an insufficient cause. In the starved condition of the patient, on account of the small amount of food absorbed, we find ample reason for his extreme feebleness.

Writers on diseases of the pancreas mention the fact that that organ is sometimes indurated aside from cancerous disease, but no explanation is given as to the cause of the hardening. In this case you will see by the report of Dr. Shakespeare that the induration seemed to arise from an inflammation that began in the ducts of the pancreas. It is very easy to see how the diminished caliber of the ducts would interfere with the exit of the pancreatic juice. Further, the inflammation extended to the surrounding tissues. More than this, the secretion of the organ, being dammed back, caused dilatation of the tubes, thus increasing their size and consistency, and interfering with the secretory process. Naturally, the function of the gland-cells is partially destroyed by obstruction to the outflow of their secretion into the intestines.

Jaundice often comes on with panc
communis choledochus may become a
the stomach causes agglutination in ne

pancreas has been found by a Brazilian physician in persons suffering with anchylostoma duodenale. (Ziemssen, p. 784, vol. vii.) This organ is sometimes affected secondarily to the stomach. In a case of dilatation of the stomach, Klohss found the pancreas atrophied.

In examining the case of this patient for the purpose of ascertaining the cause of his pancreatic disease, it is easy to suppose that the trouble, which seemed first to attack the intestines, caused an irritation of the pancreatic duct in the same manner as a like cause sometimes affects the duct of the liver. The liver is sometimes affected by the traveling of inflammation from the duodenum up to the duct of the liver, causing catarrhal inflammation of the ducts of that organ. On the other hand, it can be assumed that primary inflammation of the duct of the pancreas may have interfered with the digestion of fats, which in turn caused a fatty diarrhœa, followed by the dyspepsia. Yet the malarial element in this case may have caused the whole of the abdominal symptoms. It sometimes causes catarrhal inflammation of the ducts of the liver.

The questions of practical importance in this case are: (1) Is it possible to diagnosticate the condition of the pancreas as found in this case? and (2) What is the remedy for this disease?

In answer to the first question, I would call your attention to the anatomical relations of the pancreas. This organ is deeply seated and covered by the transverse mesocolon, the stomach, and the left lobe of the liver; behind it are the vertebræ and the large blood-vessels, all interfering with investigation. Hence the difficulty in diagnosticating a slight increase in the size of the pancreas; for you will remember that in the case here reported the increase at the two extremities of the organ was not great. The enlargement, therefore, could not be determined. The disease of the heart and stomach may have masked or added to the symptoms of this patient. With a disease as chronic as was this in its late stages, cancer could be excluded. If there had been fatty stools of years' duration, with concurrent jaundice, this would have assisted in the diagnosis.

In answer to the second question: "What is the remedy for this disease?" I would say that the experience in this case indicates that [illegible] the diarrhœa in the latter stages of the [illegible] alimentary canal is then such that [illegible] earlier stages much may be done

to prevent emaciation and the advance of this disease. Pancreatin administered with fatty food may be of service, if it is as beneficial as some claim it to be. Others, however, maintain that the gastric juice digests the pancreatin when it is administered by the mouth, thus destroying it before it can be of any service in emulsifying fats. An occasional alterative, to assist peristalsis, is useful, especially one that acts on the duodenum. Anything that would be likely to cause inflammation of the duodenum should, however, be avoided.

Dr. McCracken kindly performed for me two experiments with pancreatin. In one phial twenty grains of pancreatin were mixed with four drachms of cod-liver oil and one ounce of pure water, and resulted in an emulsion. After standing an hour, the emulsified oil settled at the bottom of the bottle. In the other phial were placed five grains of Boudault's pepsin, three drops of dilute nitromuriatic acid, and one ounce of pure water. To this was added five grains of pancreatin, and one drachm of cod-liver oil. The result of this mixture was that the oil floated on the top, unemulsified; and that a very small sediment of emulsified oil was found at the bottom after the mixture had been allowed to stand for some time. This experiment proves that when it is administered by the mouth pancreatin is rendered almost entirely inert by the juices of the stomach.

It would, perhaps, be better to order a food containing the smallest proportion of fat, the oil being introduced through the skin by frequent inunction. Much oil may be introduced into the circulation without any aid from the alimentary canal. The usual remedies for controlling a diarrhœa would then be effective. If the cure of the patient could not be effected, his life might at least be prolonged and his condition during that time rendered more comfortable.

A REPORT OF THE LARGEST RECORDED ANEURISM OF THE HEART.

By ROLAND G. CURTIN, M.D.

Cardiac aneurisms, although in themselves of little clinical importance, are on account of their rarity always interesting, and for that reason it is my desire to report the following case. I believe it to be unique, a careful search through medical literature having failed to reveal any instance in which the aneurismal sac reached the tremendous proportions of this one.

Two years before the patient came under my charge, he was admitted to the Philadelphia Hospital with general dropsical symptoms. He was under my observation for two years. During this time, and one year before his death, Dr. Pepper decided that he had a left pleural effusion, and, introducing an aspirating needle, drew off two hundred and thirty-five cubic centimetres of pure blood, which rapidly coagulated. The case was reported by Dr. Pepper as one of hæmothorax. No unpleasant symptoms followed the operation.

When the patient came under my care the whole of the left anterior chest, nearly to the axilla and clavicle, was pulsating and heaving with each beat of the heart. To the left of the sternum, at the junction of the fourth and fifth ribs, there was a circumscribed, more markedly pulsating area. He had attacks of dyspnœa on slight exertion, or when under excitement, for two years before his death, associated with a cyanosed condition of the skin, particularly about the face. He was also dyspnœic when the atmosphere was heavy.

About two months before his death, the anasarca, which had disappeared two years before, returned, with increased dyspnœa. During the last two years of his life, his radial arteries had undergone rapid degenerative changes, so that at the time of his death these vessels were like a string of beads, from their atheromatous condition. During the last few months that he lived he became weaker and weaker, and death was looked for two days before it occurred. He died rather suddenly, apparently from apnœa. At the time of death his face was greatly swollen.

Physical Examination.—On inspection, a slow, outward wave toward the left was observed, which went far out beyond the left nipple, and high up near the left clavicle. Palpation showed that this heaving, wavy impulse extended as far out as the midaxillary line, and at times beyond it. On percussion, the left side of the chest was found to be flat over its entire surface.

Auscultation showed an absence of all breath- or voice-sounds. On applying the ear to the chest to the left of the sternum at the fifth interspace, a long, rather soft, systolic murmur was heard, followed by a long, soft, whiffy sound, which cor-

responded in time with the diastole of the heart. The two sounds together occupied nearly the whole cardiac cycle.

Autopsy.—The following notes were taken at the time when the post-mortem examination was made:

"The left lung was compressed to about one-third of the size of an ordinary fist, and pushed above the position of the root of the lung, where its apex should have been. The right lung was slightly compressed, as the heart encroached upon the right cavity of the chest. Here and there were emphysematous patches. The pleural cavities contained no effusions or adhesions, and were healthy.

"The heart itself was hypertrophied, particularly the left ventricle. The aortic valves were slightly thickened and stiff. The mitral valve and the pulmonary valves were healthy.

"Springing from the left ventricle was a very large aneurismal sac, which was in close contact with the left side of the chest. The pericardium was thickened, and quite intimately adherent to the heart and the aneurismal sac, but they could be separated by the occasional use of the knife. The mediastinal pleuræ were everywhere incorporated with the pericardium. Near the base of the heart the pleura contained soft, yellow lymph, evidently the result of a recent inflammation. The aneurismal sac formed the apex of the mass; its walls were about three millimetres thick, and were composed of a dense, light, fibrous material, appearing to the naked eye to be made up of widely separated muscle-fibres, demonstrable after the pericardium was removed.

"When the sac was opened, it was found to contain a large amount of blood. In its lower part, and half-way up its sides, it was coated with laminated clots, about two centimetres in thickness. After the removal of the black, clotted blood, the cavity was filled with water. Upon weighing, its weight was found to be two thousand eight hundred and fifty grammes. On emptying the sac and reweighing it, the weight was found to be thirteen hundred and seventy grammes less. As a cubic centimetre of water weighs a gramme, it will be seen that the cavity had a capacity of thirteen hundred and seventy cubic centimetres, or nearly a litre and a half. Two holes, communicating with the left ventricle, were discovered. They were about the size of a large quill. The apertures were about a centimetre and a quarter apart, and seemed to be located in a white substance, which was undergoing atheromatous change. The inside of the sac was here aud there covered with plates of atheroma. In some places calcareous degeneration had taken place. Spicules of this material appeared in the incision, and at one place in the left side of the sac a hard plate gave forth a sharp click when struck with a knife.

"The liver was contracted, indurated, and congested. Its edges were contracted and rounded. To the right of the transverse fissure was a white spot, depressed, and looking like a cicatrix, probably an old infarction. The spleen was small, blue, and firm. The kidneys were congested, slightly contracted, and a little granular. The capsule was quite firmly adherent. There was no erosion of the vertebræ."

Realizing that the above notes, made many years ago, did not adequately describe the minute points of interest in this specimen, I asked Dr. David Riesman for a more systematic description of it, and he has most kindly furnished me with the following dictation:

"*May 26, 1901.*—The specimen before us is that of a heart which has been kept in alcohol for nineteen years. The organ is very much enlarged toward the left

side, on account of hypertrophy of the left ventricular wall and the presence of a large aneurismal sac springing from the left ventricle.

"The chamber of the left ventricle is about normal in size, but its wall is greatly thickened; in the preserved specimen it measures more than two and a half centimetres in thickness. The columnæ carneæ are well marked, and the papillary muscles very thick and strong. The mitral valve, in its anterior leaflet, presents a small sclerotic patch; it is, however, perfectly pliable. The posterior leaflet is normal. The tricuspid orifice admits three fingers. The aortic valve presents nothing abnormal, except that on one leaflet, at its insertion into the aorta, there is a small, elevated ridge of atheroma on the ventricular surface. The aorta is of normal calibre, and has, scattered through it, a few calcified plates. The coronary openings are patulous, although one is rather small.

"In a somewhat funnel-shaped recess, situated between the two sets of papillary muscles of the posterior mitral leaflet, are two oval openings connecting the aneurism with the ventricular cavity. One opening has its long axis transversely, and measures about one centimetre in length and three millimetres in width; the second is of about the same dimensions as the first. The margins of both openings are smooth, and have a glistening, fibroid appearance. This is particularly true of the inner opening. On the ventricular surface these openings are separated by a bridge of tissue, six millimetres wide and one centimetre long. On the aneurismal surface the openings are large, somewhat funnel-shaped, and surrounded by fibrous tissue, slightly puckered. The outer opening is undermined, and communicates with the ventricle by a third perforation, opening about eight millimetres to the outer side of the median line, in a recess formed between the corresponding papillary muscles belonging to the anterior and posterior mitral leaflets. This opening on the ventricular surface is oval, with its long diameter vertical to the wall of the ventricle, and measures one centimetre by three millimetres. There are thus three opening on the ventricular surface and two on the aneurismal. They are all situated about three and three-fourths centimetres from the auriculoventricular ring.

"The aneurism itself is the size of a child's head, and springs from a point almost opposite the ventricular septum. It has thick, tough, fibrous walls, that can be separated into at least three layers. When not covered by clot, the internal surface is quite smooth and glistening. There are thick, laminated clots at the bottom of the sac, around its attachment to the ventricle, particularly at a point corresponding with the apex of the ventricle. The sac has been incised by a long, vertical cut, and shows no point of rupture. There are some signs of old adhesions on the external surface, especially at the base of the attachment of the sac to the wall of the heart. At its base the sac measures fifteen centimetres vertically, and thirteen and three-quarters centimetres transversely, while from the ventricular wall externally to the inner wall of the sac the distance is sixteen and a quarter centimetres. To the outside of the openings is a crescentic, projecting ridge, forming a niche about two and a half centimetres deep and five centimetres high. This ridge is continued below as a low elevation, for a distance of five centimetres, to a point two and a half centimetres below the openings.

"The chamber of the right ventricle is not enlarged. The wall is slightly thick, and measures about one centimetre in diameter. The right auricle presents nothing abnormal. The pulmonary and tricuspid valves are normal. The foramen ovale is closed. The left auricle is normal.

"As the heart is now, it having been somewhat flattened in the jar, it would appear that the apex was in a large degree constituted by aneurism."

There are three points of interest in this case:

(1) So far as I am able to learn, it is the largest aneurism of the heart that has ever been recorded.

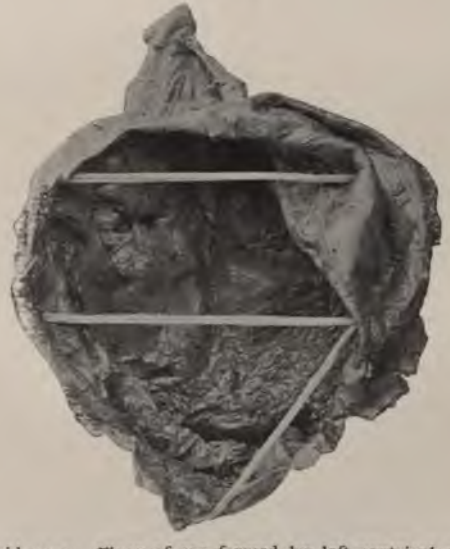
(2) The patient lived for years, not only with greatly diminished breathing capacity, but also with a severe complicating cardiac condition.

(3) The removal of two hundred and thirty-five cubic centimetres of blood, to accomplish which must have caused a break in the continuity of the aneurismal walls, failed to produce any symptoms whatever.

FIG. 1.

Posterior view of heart showing aneurismal sac distended. The line *a b* shows the boundary between left and right ventricles.

FIG. 2.



Aneurismal sac laid open. Floor of sac formed by left ventricular wall two and a half centimetres thick. Capacity of sac, thirteen hundred and seventy cubic centimetres; length fifteen centimetres; breadth, thirteen and three-quarters centimetres; depth, sixteen and one-quarter centimetres. (Both illustrations are much reduced in size.)

A CASE OF INTERVENTRICULAR ANEURISM OPENING INTO THE RIGHT VENTRICLE BY ULCERATIVE PROCESS, AND ANEURISM OF THE ABDOMINAL AORTA, WITH AUTOPSY.

By ROLAND G. CURTIN, M.D.

"*History. January 9, 1885.*—Matthew D., aged fifty-three years; single; white; born in Ireland; came to America when three years old.

"*Family History.*—Mother and father died of old age. Has had three brothers and one sister. One brother died of typhoid fever, one was shot during the War of the Rebellion, and the third died of an unknown cause. The sister is alive and well.

"*Past Medical History.*—Has used whiskey and beer to excess, and tobacco moderately, all his life. Has always had fairly good health. Had measles when a child, and chills and fever six years ago. Has never had rheumatism.

"In 1865 he had a primary sore, which made its appearance about nine days after exposure, and was followed by no secondary symptoms. Five years ago he had a severe fall from a horse. He first noticed palpitation of the heart and shortness of breath three years ago, but considered himself perfectly well, except for some precordial pain, until March, 1884. At that time he was seized with an attack in which he became very faint, fell down unconscious, and remained so for several minutes. On attempting to get up, some three hours later, he found himself still very weak. He did not have another attack until March, 1885. This attack lasted about an hour, with a period of unconsciousness of probably ten minutes. At this time he vomited, but on no occasion did he expectorate any blood.

"Since the date just mentioned, he had a number of attacks, which seemed to be becoming more frequent, although they were apparently not more severe than at first.

"*Physical Examination.*—The precordia is prominent and bulging. There is a very apparent area of pulsation, five centimetres in diameter, in the third interspace, between the mammary line and the left border of the sternum. The apex beat is visible within the nipple line, and extends over an area which would correspond to a circle with a diameter of three and three-fourths centimetres.

"Over the pulsating region, as above given, is heard a systolic murmur, most intense over and above the second costal cartilage, at a point midway between the nipple-line and the left border of the sternum. Palpation shows there is also present a marked thrill.

"The murmur is transmitted over the entire precordia and into the great vessels of the neck. It is also heard in the left interscapular and axillary spaces and at the angle of the scapula on the same side. Over its seat of maximum intensity this murmur can be heard with the ear removed from the chest.

"The patient also complains of a sharp pain shooting through to the back,

which, from the murmur also present here, probably has its origin in the abdominal aorta."

Note, November 20, 1885.—"The patient is now suffering from one of his attacks. He feels faint, and his whole body vibrates with each systole of the heart. The pulse is 136, gaseous, small, and feeble. He states that at the beginning and close of his attacks his eyesight fails and his eyeballs become more prominent."

Note, December 3, 1885.—"Posteriorly the point where the murmur is heard with greatest intensity is on the left side, opposite to and five centimetres to the left of the second dorsal vertebra. It is transmitted five centimetres father to the left, gradually growing fainter, but being still audible at the point of the shoulder. Anteriorly the murmur is most intense in the second intercostal space, two and a half centimetres to the left of the sternum, where it may be heard with the ear five centimetres from the chest.

"Associated with this murmur there is an area of dulness, over which may be felt a thrill, extending from two and a half centimetres below the clavicle to the lower border of the third rib, and from the right edge of the sternum to a point just in front of the anterior border of the left axilla. With the palm of the hand placed upon this area, the left arm extended, and used as a stethoscope, the murmur may be heard by another placing his ear at the left elbow-joint."

"*Autopsy, March 26, 1886.*[1]—Left thorax prominent up to the upper border of the second rib, extending over the entire heart region. Pericardial sac bulging. Precordial space full, and entirely uncovered by lung. Thoracic aorta slightly dilated; there is a small, saccular dilatation above its origin anteriorly, capable of admitting a small chestnut. Inner surface roughened, with some calcified plates on the arch.

"*Heart.*—Left auricle much dilated; mitral orifice dilated, admitting more than three fingers. Valves slightly thickened. On opening the right ventricle, an aneurism of the undefended portion of the interventricular septum is found bulging into the right ventricle, into which it opens through a small orifice, probably the result of an old ulceration. This aneurism is large enough to contain a good-sized walnut. The right auricle is also dilated.

"The liver and kidneys show considerable passive congestion.

"The abdominal aorta is much thinned at its commencement, and throughout its entire length is the seat of extensive atheromatous changes. Extending from the diaphragm to the iliac arteries is a fusiform aneurism filled with old coagula, its lumen being occuped by recent clots. There is no erosion of the vertebræ."

[1] All notes made at autopsy and not directly bearing upon the physical signs are omitted.

AMYOTROPHIC LATERAL SCLEROSIS.

By CHARLES K. MILLS, M.D.

Amyotrophic lateral sclerosis is a disease that has for its chief symptom progressive muscular atrophy, fibrillary tremor, contracture, exaggerated deep reflexes, and in some cases progressive bulbar symptoms, sensory manifestations being absent. Pathologically it is characterized by progressive degeneration attacking simultaneously or successively both the lower and the upper segments of the motor path, although the relative degrees with which these portions of the motor system are invaded differ greatly in different cases. By Gowers, and some other authorities, amyotrophic lateral sclerosis is made practically synonymous with the diseases usually described as chronic muscular wasting, progressive muscular atrophy, chronic poliomyelitis, and wasting palsy. It is best, however, to make separate uses of the terms amyotrophic lateral sclerosis and progressive muscular atrophy, the latter to indicate that form of slowly progressive muscular degeneration which has for its pathological characteristics degenerative lesion of the lowest motor segment, the so-called myelopathic or Aran-Duchenne type of muscular atrophy; while the former name is applied to that form of chronic muscular wasting associated with spastic conditions and exaggerated reflexes which is due to involvement of both the motor pyramidal system and the peripheral motor system. The disease has been described under the name of spastic spinal paralysis with muscular atrophy.

The history of our knowledge of the development of amyotrophic lateral sclerosis is not ancient. Our first exact knowledge regarding the disease dates back to Charcot's descriptions in 1869 and later. Among the most valuable articles in text-books and treatises are those of Gowers and of Beevor. The most important American contributions are those of Collins, of Dercum and Spiller, and of Spiller. Wolff, in 1894, collected ninety cases, and Collins, in 1896, succeeded in collecting seventy-two cases with necropsy. In the papers and treatises here alluded to will be found references to all the literature of value.

Several varieties of amyotrophic lateral sclerosis can be recognized, these being based upon peculiarities in symptomatology and on special associations. Thus, Gowers describes three varieties in his discussion of chronic muscular wasting: (1) The atonic variety, in which both arms and legs are the seat of wasting without spasm; (2) muscular weakness with spasm, but without atrophy, or only slight atrophy; (3) tonic atrophy, in which both wasting and spasm are present, but rarely extreme. According to the views held by the writer, the purely atonic variety of atrophy should not be included under amyotrophic lateral sclerosis, except in particular cases, which are to be explained by the fact that if the lower motor neuron segment degenerates first, and especially if the degeneration is complete, the conditions produced may afterward mask the existence of degeneration in the corticopyramidal system. In considering the muscular atrophies of spinal origin, Charcot divided them into the protopathic and deuteropathic, classing under the former those cases due solely to degeneration of the cell bodies in the ventral horns, and, under the latter, cases of amyotrophic lateral sclerosis, to which he gave the name dueteropathic, because he supposed the degeneration of the peripheral motor neuron system was secondary to that which occurred in the pyramidal tract. We believe it preferable to regard the two forms of disease as separate entities, although it is undoubtedly true that in many cases of apparently pure protopathic type both motor neuron systems are in some degree implicated. Varieties based upon special combinations of the amyotrophic syndrome with single symptoms or symptom groups of other well-known degenerative affections are found in practice. Bulbar amyotrophic sclerosis may be associated with disseminated sclerosis irregularly distributed through the neuraxis. Tabes and amyotrophic sclerosis are a not unusual combination.

While the symptomatology of amyotrophic lateral sclerosis presents many irregularities and departures from a common type, the majority of cases have a method of onset, progress, and termination and a symptomatology which clearly indicate the affection. The disease is usually of comparatively rapid progress, many of the patients dying within three or four years of the time that the initial symptoms are observed, but in a few instances, like tabes and disseminated sclerosis, it is much prolonged. Commonly the first symptoms are slight atrophy, with equally slight muscular weakness and spastic tendencies in the upper

extremities. Fibrillary tremor may soon be present in some of the wasting muscles, the symptoms presented by the upper extremities often having advanced considerably before other parts of the body are involved. But sooner or later, and in some instances early, the patient complains of weakness in both lower extremities. Still later a group or a series of bulbar manifestations are added to the syndrome. The first wasting may be in the interossei, in the muscles of the forearm, in the deltoids, or in some of the muscles of the back or front of the trunk, or several of these regions may be almost simultaneously attacked. Muscular wasting in the lower extremities is often not very evident until a comparatively late period. The bulbar symptoms are usually those related to the cranial nerves, from the seventh to the twelfth, although in rare cases the ocular muscles may be implicated. These bulbar symptoms are in the main those described under the clinical designation of *glossolabilaryngophayngeal* paralysis—that is, paresis with wasting of lingual, oral, facial, laryngeal, and pharyngeal muscles, with the well-known accompanying train of symptoms. At an early period examination of the patient shows exaggerated deep reflexes, quadriceps, gastrocnemius, patellar, Achillic, biceps, triceps, scapular, and other reflexes are or become much exaggerated. The Babinski phenomenon may be present, contractures set in, and as the diseases descends to its fatal termination, threatening cardiac and respiratory phenomena are added. On the negative side all forms of sensory disturbances are absent, and usually the patient retains his mental powers until a late period, or to the last, except in so far as they are obtunded by his suffering and by his general malnutrition.

It is usually stated that the disease begins and shows its first advancement in the upper extremities, and this is doubtless true for many and probably for most cases, but in some instances the upper extremities are involved after the lower, or the implication may even be preceded by bulbar symptoms. In the case recorded by Dercum and Spiller, the weakness and marked spasticity of the lower extremities, with exaggeration of the reflexes were observed for at least two or three years before the upper extremities were affected. It is probable that slight involvement of the upper extremities may escape notice for a time, but where the patient has maintained good use of his arms long after spasticity and paresis in the lower extremities have attracted attention

through interference with station and gait, the prior implication of the lower extremities cannot be doubted.

The existence of some degree of spasticity is a cardinal feature of amyotrophic lateral sclerosis, except in those cases to which allusion has been made in which the atrophy advances to a marked degree before the lateral columns are affected. In the earliest period of those cases which Charcot regarded as representing the type of amyotrophic lateral sclerosis as distinguished from protopathic muscular atrophy, the patient complains of weakness and painful or distressing sensations in the upper extremity, with some stiffness or spasticity. This increases even to the point of a more or less marked rigidity, the arms wasting, but not at first presenting an atrophic appearance. A few months later the limbs become spastic, but without marked wasting. The conditions in both upper and lower extremities are much like those which have been described as present at an early stage in the rare cases of primary lateral sclerosis. Increased myotatic irritability is coincident with the development of spasticity. Later and irregularly, as regards time and location, marked muscular atrophy develops, especially in the upper extremities and in the trunk. In other cases true atrophy advances step by step with spasticity and contracture, or atonic atrophy may precede for some time the development of contracture. In comparatively rare cases the facial and other muscles supplied by the cranial nerves are hypertonic, even a certain degree of trismus being present.

Much space might be taken up by a description of the various groups of muscles which undergo atrophy, and some attempt might be made to indicate an order in which this atrophy most frequently occurs. A study of many cases however shows that the musculature degenerates in a very irregular fashion, both as regards location and time. The description of the atrophic conditions present in progressive muscular atrophy of the myelopathic type would perhaps answer in many of the cases of amyotrophic lateral sclerosis. The adductor muscles of the thumb, thenar, and hypothenar eminences, the muscles of the shoulder girdle, the pectorals, the glutei, the peroneal muscles, the tongue; in late stages the diaphragm and internal muscles of respiration, and even the visceral muscles become involved. Almost any muscle or muscular group may be attacked as the disease advances. On the whole it might perhaps be said that in amyo-

trophic cases some preference is shown in the process of degeneration for the muscles of the hand and forearm, and when the distal portions of the upper extremities show atrophy with contractures and hypertonicity without objective anesthesias amyotrophic lateral sclerosis may usually be confidently asserted.

In at least a few cases which ultimately become clear instances of amyotrophic lateral sclerosis, no muscular atrophy is evident for periods varying from one to seven years. These are the cases which seem to support the view of Charcot that a distinction should be made between amyotrophic lateral sclerosis and protopathic progressive muscular atrophy. They are also, in at least some instances, the cases which are regarded for some time, and it may be for years, as examples of pure lateral sclerosis. I have followed several such cases for periods of from five to ten or more years, usually the observations being interrupted, but occasionally, as at the Philadelphia Hospital, they have been continuous. In order to illustrate the fact alluded to above, namely, that spasticity even to the extent of rigidity often precedes any atrophy by a long interval, Beevor relates the case of a young woman who first became weak and stiff in the lower extremities, a year later in the upper, and who showed moderate atrophy in the lips and tongue at the expiration of two years. At this time rigidity had become general, and she had marked exaggeration of all the reflexes, even including those of the jaw and face. I do not see how it is possible in the light of such cases, of which I have seen a number, to give adherence to the view of Leyden, Gowers, and others, that no real distinction needs to be made between progressive muscular atrophy of the protopathic type and the disease under consideration. It is undoubtedly true that one of these forms of degeneration often merges into the other, and that degeneration of the ventral cornua may precede by a definite interval the sclerosis of the pyramidal system, but it is also proved that the degeneration of the peripheral motor neuron system may exist separately, and both from the clinical and the pathological stand-point may never be associated with degeneration of the upper motor segment.

In the early stages of a case beginning with degeneration of the upper motor segment, responses to the electrical current are little if at all changed. As the case advances quantitative diminution at first occurs, and as atrophy proceeds impaired response both to faradism and galvanism is observed. With impaired or even

lost faradocontractility the galvanic response may remain, and may present in an irregular fashion the phenomena of degeneration reaction. In advanced cases both modal and serial changes are readily observed in some of the muscles affected. When the muscle is both spastic and moderately atrophic, a more or less tetanic response may be produced by the application of the galvanic current. In brief, the electrical responses are changed in proportion to the extent and degree of the central and the muscular degeneration.

Even before the disease has made marked advance the deep reflexes are markedly increased. Tendon and muscle jerks are exaggerated, phenomena which can be elicited in the upper as well as in the lower extremities. Biceps, triceps, and wrist jerks are prompt or even much exaggerated. Myotatic irritability is pronounced. The Von Bechterew scapular reflex is usually present. Knee jerks are greatly increased, and both knee clonus and ankle clonus may usually be elicited. At some period the Babinski reflex becomes evident. Even the deep reflexes of the lower jaw may be obtained—the so-called chin jerk, or jaw jerk, to which attention was first called by Beevor and by Morris Lewis. In 1881 Beevor observed spontaneous clonus of the lower jaw in the case of a woman suffering from amyotrophic lateral sclerosis. This response is obtained by tapping the finger or a pleximeter placed along the lower jaw, the mouth being held slightly open; this causes a contraction or series of contractions of the masseters, snapping the jaw upward or even causing a clonus of the lower jaw, phenomena which seem to show that the pyramidal tracts above the cranial nuclei are involved in the degenerative process.

In the majority of reported cases, and in all cases observed by the author, sensory symptoms have been absent, but this is not the invariable rule. In a very few cases some sensory symptoms have been present, and some have recorded these cases with sensory symptoms as constituting a link between amyotrophic lateral sclerosis and progressive muscular degeneration on the one hand, and tabes on the other (Hektoen). Primary degeneration of the columns of Goll has been found in several cases, but this degeneration is not the same as that observed in true tabes, and in these cases the dorsal roots have been found diseased. It follows that some other explanation of the degeneration of Goll's columns must be

sought for than that which accounts for the degeneration in cases of tabes. Tabes and amyotrophic lateral sclerosis may exist in the same case, and such a case has been recorded by me.

Most writers on the subject of amytrophic lateral sclerosis lay little or no stress on the presence of mental symptoms, and some say they are absent. It is probable that little mental change takes place until late in the disease, but in one of my cases the patient showed decided deterioration during the last months of his life. Spiller found some mental deterioration in connection with the case in which he studied the cerebral cortex, as well as the entire motor neuron systems. Disturbance of mentality was present in a case recorded by Probst, and was attributed by him to the changes in the cortex; and Sarbo, who has reported a case with predominating mental symptoms, found changes in the tangential fibres, and fatty granular cells in the central convolutions. Spiller found changes similar to those determined by Sarbo, not only in the motor region, but in various districts of the cerebral cortex, and he believed that the widespread cellular pigmentation may have been the cause of the mental deterioration.

While a case of amyotrophic lateral sclerosis may pass through its entire history without any evidences of involvement of the bulbar nuclei and pyramidal tracts above these nuclei, the implication of the bulb is very common in the experience of the writer. In a case in my service at the Philadelphia Hospital, which has been recorded by Spiller in the paper frequently referred to in this article, bulbar symptoms were among the earliest, and they were throughout among the most pronounced phenomena of the case. In the case of Dercum and Spiller the bulb was early attacked, probably before the cervical cord. The motor nuclei and the portions of the pyramidal tracts supplying these nuclei are chiefly attacked. Unilateral and bilateral atrophy of the tongue, atrophy of the trigeminal musculature, of the facial muscles, and even of the muscles of the pharynx and larynx, may be present and in consequence movements of the face and jaw and disorders of articulation, deglutition, and even of phonation are present. Involuntary laughing and crying have been noted in several cases, just as the same phenomena are observed in disseminated sclerosis without nuclear symptoms. Schlesinger has reported a most interesting case with bulbar symptoms. A

man, seventy-two years old, received a severe mental shock in the loss of his position, and his speech became at once affected. Soon after this a temporary right-sided hemiparesis occurred. The mouth was opened with difficulty, and deglutition was affected. The secretion of saliva was increased, and forced laughter and crying were noticed. Tremor of the head and limbs like that of paralysis agitans developed. The pupils did not react to light, and only slowly to convergence and accommodation. To this bulbar palsy were added progressive spastic paresis with distinct atrophy of the extremities, and finally spasm of the vesical sphincter and great exaggeration of all tendon reflexes. The necropsy showed that the case was one of amyotrophic lateral sclerosis. An acute commencement, or a rapid progression of bulbar palsy should always suggest the possibility of amyotrophic lateral sclerosis. In fifty-eight cases collected by Probst, and cited by Spiller, the first symptoms were bulbar in nine. In most of the cases in which bulbar symptoms were present at all, they appeared either first or soon after the involvement of the extremities; in those cases in which the bulbar symptoms were the first signs, the extremities were soon involved. Sometimes the bulbar symptoms did not appear for one or even two years after the extremities were attacked, and in a few cases they never appeared.

Typical cases of amyotrophic lateral sclerosis pass slowly, or it may be with comparative rapidity (in a period of three or four years), through the imperfectly demarcated stages which have been indicated in the preceding general and detailed symptomatology of the disease—through the stage of discomfort, weakness, and early spasticity with increasing reflexes, and through that of tonic atrophy, with intensification of preceding active phenomena, finally reaching a terminal period in which the patient usually presents atrophy extreme and generalized, rigidity and deforming contractures, and in the majority of instances marked bulbar symptoms. As has already been shown, types of amyotrophic lateral sclerosis are observed in which bulbar symptoms may precede the involvement of either the upper or lower extremities, may be the earliest manifestations, the symptoms in the extremities following, or they may indeed be the only manifestations, giving in this restricted form a true bulbar paralysis; in addition it must be borne in mind that the most frequent termination of all cases is in bulbar involvement, and when death does not

ensue from intercurrent cause, the fatal issue is determined by the slow destruction of the nuclei of those cranial nerves the integrity of which is absolutely essential to the prolongation of life. In the last period of the disease therefore in addition to atrophy and contractures, the trunk and limbs present various trophic and other lesions, some primary and some secondary. Bed-sores, changes in the nails, cutaneous eruptions, and involuntary evacuations may be present, although some authors state (what is not in accord with my own experience) that the bowels and bladder are usually unaffected to the last. Articulation becomes more and more indistinct; even vocalization may be affected; deglutition becomes imperfect, or impossible except artificially; respiratory and cardiac action is first simply disturbed, then intermittently interfered with or arrested, death sometimes taking place through a paralysis of heart or lungs, undoubtedly the result of destructive degeneration of the pneumogastric nucleus, or at least of the motor portion of this nucleus.

The etiology of amyotrophic lateral sclerosis is as obscure as is that of all other forms of sclerosis except tabes dorsalis. Most writers give as causes traumatism, exposure to cold and wet, physical or mental strain, sexual excesses, and other forms of dissipation, just as they give the same causes for other diseases, degenerative and otherwise, about the etiology of which they are lacking in exact knowledge. Any set of twenty or more cases can be taken, and causes such as this found for a greater or less percentage of them. Probst, for instance, found that with regard to his fifty-eight cases, in seventeen the disease was attributed to cold, in four to psychic trauma, in three it began during pregnancy, in two the patients had had syphillis, in one the hygienic surroundings had been bad, in three trauma was assigned as the causes. It is altogether probable that a neuropathic tendency is one if not the most important etiological factor. Syphilis does not seem to play an important rôle, although a preceding history of syphilis was present in two of my cases, but in these alcohol and other excesses were also a part of the history. It is probable that in one with a neuropathic predisposition, any cause which will act powerfully upon the central nervous system, such as infection or toxine, traumatism or excessive exposure may induce the disease. Statistics would seem to indicate that the disease occurs almost as frequently in one sex as in the other, although Charcot and Marie believe that females are

more liable to it than males. It comes on most frequently between the ages of thirty and sixty, but may be regarded as occurring at all periods. Probably the most susceptible decade is from thirty-five to forty-five years.

In a typical case of amyotrophic lateral sclerosis which has lasted for several years, and which has shown the well-known symptoms and signs of the disease in the extremities, trunk, and parts supplied by the motor cranial nerves, marked degenerations will be found in all portions of the upper and lower segments of the motor systems of neurons, and in the nerves and muscles supplied from these segments. Let us recall here the now well-known fact of the separate existence and the extensions of these two segments. The upper motor neuron segment begins in the cell bodies which are spread out over the motor region of the cortex, that is, chiefly in the precentral, postcentral and paracentral convolutions, and in the posterior extremities of the first, second, and third frontal convolutions. The axons which emerge from these cell corpora pass downward in the pyramidal tract to the nuclei of the motor cranial nerves, and also to the motor nuclei situated at all heights in the ventral horns of the spinal cord. Here they terminate by arborizations which surround and touch the cell bodies of the cranial nuclei and the ventral horns, but do not become anatomically continuous with these structures. The lower motor neuron segments begin in the cranial motor cell nests, in the pons and oblongata, and in the ventral horns, these sending out axons which pass to the muscles. When both these motor segments are attacked in amyotrophic sclerosis one process is not consecutive to the other, or at least does not depend upon the other, but it is probable that both are attacked independently, sometimes coincidently, and sometimes at different periods. Usually the pyramidial systems are attacked first. Pathological examination will therefore show degeneration of the cell bodies in the motor cortex and of the pyramidal systems of fibres in cerebrum, crus, bulb, and cord. This degeneration has been much more frequently traced in the cord than in the parts higher up, although its presence would be theoretically expected in greater or less degree at all heights. Degeneration and atrophy are also present in the nuclei of the motor cranial nerves, and in the cell bodies of the gray cornua, the peripheral motor nerves and the muscles supplied by them also being atrophic. A few careful investigators have searched all parts of

the nervous system for the signs of disease and degeneration, the result being that they have found this restricted in the main to those parts which in one way or another are related to the musculature of the body. In exceptional cases, however, other parts have been found degenerated, as the dorsomedian columns of Goll. All the findings in the lower motor neurons and their musculature are the same in this disease as in the pure myelopathic type of progressive muscular atrophy. That which distinguishes this disease pathologically from this form of progressive muscular atrophy is the existence of chronic degenerative lesions in the upper motor segment. I shall now give in detail the lesions found in this disease, the description being based chiefly on the pathological studies made by Spiller in two cases already referred to in this article. I had the opportunity of studying both of these cases during life, one of them having been in my own service in the Philadelphia Hospital, and the other in the service of my colleague, Dr. Dercum. While many investigations have been made in cases of amyotrophic lateral sclerosis the majority of these have included studies which extended only over comparatively limited areas of the central nervous system. The gray and white matter of the cord were the first to be studied with accuracy and thoroughness. In many cases efforts were not made to trace degeneration above the decussation; in many cases also the examinations were not extended to tissues outside of the nervous system, as to the muscles, muscle spindles, and bones. Spiller gives the literature of the subject so fully as to make it necessary only to refer to his monograph. Including Spiller's case, up to the time of the publication of his paper in 1900, the degeneration of cell bodies and fibres had been traced from the periphery to the motor cortex in only ten cases, and the completeness of the investigations in these cases differs considerably. He not only traced the degeneration to the cortex, but in common with only one or two others studied the lesions present in the callosum. In the case reported by Dercum and Spiller there was marked degeneration of the dorsal columns in the cervical region, but this was not found in Spiller's subsequently reported case. Other reporters have described notable degeneration of these columns in all portions of the cord. Different degrees of degeneration have been found in Clarke's column, and the direct cerebellar has sometimes been degenerated. Degeneration of the cell groups of the ventral horns is marked, and is usually seen in

higher degree in the inner groups of cells. In several of the reported cases, noticeably in those of Mott and of Spiller, the cortex has been well studied. These studies point to degeneration of the cell bodies and fibres over almost the entire motor cortex when the investigations have been thoroughly conducted. In other regions of the cortex, as in the prefrontal, temporal and occipital lobes, pigmentation of the cell bodies is found, but not the marked degeneration found in the motor areas. The centrum ovale of the non-motor regions has been shown to be free from degeneration in all recorded cases, while in that part related to the motor cortex degeneration is advanced. Spiller was able, by an elaborate study of the cerebral hemispheres, to map out the cortical motor area, a new method of making this anatomicophysiological differentiation. The large pyramidal cells are less numerous, and many of those which remain are pigmented and atrophied. Tangential fibres are degenerated. In some cases, as in the one recorded by Mott, degeneration of the central convolution is so marked as to be observable by the naked eye when the hemisphere is inspected. In the case of Spiller, and in a few other reported cases of amyotrophic lateral sclerosis, the degeneration of the internal capsule was carefully studied, and it is interesting to know that the reports of these investigations practically coincide in giving the middle region of the posterior limb of the internal capsule as the part degenerated, a fact which is hard to explain unless it indicates that it is only in this portion of the capsule that fibres concerned with the movements of the extremities, especially of the upper extremities, are located. Mellus, in his experiments on the monkey brain, found a similar limitation of the degeneration in those cases in which he operated on the facial, the leg, and the arm centres. Probst and Spiller have reported intense degeneratian of numerous fibres in the callosum, another confirmation of the view that the callosum is directly concerned with motor functions, that it forms a part of the motor system. In several of the cases carefully studied, degeneration of certain of the motor nuclei and fibres arising from them was noted, especially in the hypoglossal, dorsal nucleus of the vagus, and nucleus ambiguus. The facial nucleus has sometimes been found degenerated, at others almost normal; the nuclei of the ocular nerves have been degenerated in one or two cases, but frequently they have been normal, these findings being in consonance with the fact that ophthalmoplegias may be present

in the disease, but are rare. Spiller detected marked degeneration in the fibres of the ulnar nerve side by side however with undegenerated fibres. Various muscles of the upper extremities showed degenerated fibres often in close proximity to fibres entirely normal. The recurrent laryngeal nerve and the laryngeal muscles were not certainly degenerated in the case of Spiller. It is probable that in other cases with more marked laryngeal symptoms such degeneration might be found. The muscle fibres of the tongue are frequently the seat of marked degeneration. The intramuscular nerve fibres and muscle spindles have proved to be normal in the cases investigated.

The diagnosis of amyotrophic lateral sclerosis is usually readily made, but occasionally, and especially when the disease is complicated with the phenomena of other degenerative affections, or with those of a focal lesion, the diagnosis may for a time present some difficulties. It may also be doubtful in the atonic atrophic variety, where the peripheral motor neuron system has degenerated before the corticopyramidal system, and consequently some of the symptoms of the latter degeneration remain masked. The chief affections from which amyotrophic lateral sclerosis needs to be differentiated are progressive muscular degeneration, disseminated sclerosis, symmetrical lateral sclerosis (if this disease has an existence), bulbar and spinal poliomyelitis, cortical encephalitis of Strümpell, cervical hypertrophic pachymeningitis or any other focal variety of spinal pachymeningitis, syringomyelia, the muscular dystrophies, and possibly multiple neuritis and diffuse myositis. The clinical differentiation from progressive muscular atrophy is to be made by a consideration of the fact that in amyotrophic lateral sclerosis, tonicity or spasticity with contracture and markedly exaggerated reflexes are present. The other symptoms are much the same. Bulbar phenomena are more frequent in amyotrophic sclerosis, and usually the disease comes on more rapidly and runs its course in a shorter time. From disseminated sclerosis the diagnosis is to be made chiefly by the presence of atrophy, by the fact that the amyotrophic disease usually begins in the upper extremities, and by the absence of the intention tremor, peculiar speech disorders, and nystagmus of disseminated sclerosis. Occasionally the two diseases merge into each other and give a confusing picture. It is probable that a fair percentage of cases set down as lateral sclerosis have been cases of amyotrophic lateral sclerosis in a com-

paratively early period. The chief features which distinguish symmetrical lateral sclerosis from amyotrophic sclerosis are the more usual beginning of the former disease in the lower extremities and the presence in it of great spasticity without atrophy. As amyotrophic lateral sclerosis is frequently to a marked degree a bulbar disease, the other forms of bulbar disease may sometimes present difficulties of diagnosis. The polioencephalitis superior and inferior of Wernicke are however diseases of acute onset, and their manifestations are confined to the bulb. They have not the features of a progressive degenerative disease in their mode of origin, and the extremities do not exhibit clinical phenomena. Of Strümpell's cortical encephalitis it need only be said that this disease is also of acute onset, generally unilateral and could only present an indistinct resemblance to an unusual type of amyotrophic lateral sclerosis. Spinal poliomyelitis is again a disease of acute febrile onset of restricted residual phenomena; the resulting paralysis and atrophy in poliomyelitis are usually limited to certain muscular groups. The muscles present complete degeneration reaction, and the muscles affected are the seat of marked vasomotor phenomena, as lowered temperature and pallor. In cervical hypertrophic pachymeningitis, atrophy with contracture may be present on one or both sides, but the disease differs from amyotrophic sclerosis, beginning in the upper extremities, in the absence of subjective and objective sensory phenomena, in the presence of complete degeneration reactions, and in the more limited distribution of the neuromuscular phenomena. The combined sensory motor syndrome of syringomyelia—atrophy with dissociated anesthesia—usually serves to separate this disease from the amyotrophic lateral sclerosis, which in nearly all cases is a purely motor disease. Multiple neuritis could not be mistaken for amyotrophic lateral sclerosis if a careful examination of the history and present state of the patient were made. Even if both upper and lower extremities were atrophied as the result of a remote attack of multiple neuritis, the absence of fibrillary tremor, spasticity, and exaggerated reflexes in multiple neuritis would be sufficient for the purposes of differentiation. In multiple neuritis, as is well known, it is usual to have diminished or lost tendon reflexes in the limbs affected. Finally, spinal or cerebrospinal syphilis may present difficulties of diagnosis in particular cases. The points of similarity are the presence in both diseases of more or less spastic

paresis; in certain types of syphilis the presence of exaggerated deep reflexes; in certain other types the presence of bulbar or, more properly pseudobulbar symptoms; and the existence in both diseases of symptoms referable to lesions both of the intracranial and intraspinal portion of the cerebrospinal axis. A little attention however to the mode of onset and progression of a case of amyotrophic lateral sclerosis, as suggested in the general symptomatology, would be sufficient to make the diagnosis clear.

The prognosis is bad, the disease usually terminating fatally in a few years. Rest and nutrient tonics offer the only hope of delaying the downward march of the disease.

BIBLIOGRAPHY.

Gowers, W. R., A Manual of Diseases of the Nervous System, 2d ed., vol. i., Phila., 1899.
Charcot, J. M., Leçons sur les maladies du système nerveux, 2d s., Par., 1869.
Beevor, C. E., In A System of Medicine by Many Writers, ed. by T. C. Albutt, vol. viii., N. Y., 1899.
Collins, J., American Journal of the Medical Sciences, 1896.
Dercum, F. X., and Spiller, W. G., Jour. Nerv. and Ment. Dis., vol. xxvi., 1899.
Wolff, Zeitschrift f. klin. Med., 25, 1894.
Leyden, E., Zeitschrift f. klin. Med., 21, 1892.
Hektoen, L., Jour. Nerv. and Ment. Dis., 1895.
Spiller, W. G., Contributions from The William Pepper Laboratory of Clinical Medicine, Phila., 1900.
Probst, Arch. f. Psychiatrie, vol. xxx., p. 766.
Sarbo, Deutsche Zeitschr. f. Nervenheilk., vol. xiii., Nos. 3 and 4.
Charcot, J. M., and Marie, P., Arch. de Neurol., vol. x., 1885,
Mott, F. W., Brain, Lond., vol. xviii., 1895.
Mellus, E., Jour. Nerv. and Ment. Dis., April, 1899.

GENERAL SARCOMATOSIS; INVOLVEMENT OF THE GENERAL NERVOUS SYSTEM.

By F. X. DERCUM, M.D.

PROFESSOR OF NERVOUS AND MENTAL DISEASES, JEFFERSON MEDICAL COLLEGE.

The following case, because of the involvement of the nervous system, and especially because of the mode of onset of the symptoms, is of unusual interest:

J. G.; white; aged fifty-four; birth-place, England; occupation, bricklayer. Admitted to the Philadelphia Hospital November 20, 1902.

Family History.—Father and mother dead; cause unknown. One brother and four sisters living, their present condition being unknown to the patient; has four sons and one daughter, all living and well. No tubercular or malignant disease in family.

Personal History.—Patient has no knowledge of having had any diseases in childhood, and claims that he never had any illness severe enough to confine him to bed until eight years ago, when he had an attack of malarial fever, lasting about three weeks. Since then he was perfectly well, until eight weeks ago, when he had a sensation of numbness and tingling in the left leg, and afterward in the right leg and arm. Three days later he fell while walking on the street, and was carried home. He was not unconscious at the time, but there was complete loss of power of both lower extremities. There was also retention of urine and incontinence of fæces. His appetite was much impaired, and he slept poorly.

Examination on Admission. General Appearance.—A large, poorly-nourished man. Has been a heavy drinker. Admits a specific history.

Tongue.—Coated, fissured, and slightly tremulous.

Chest.—Elongated, intercostal spaces visible; sternum and costal cartilages prominent.

Heart.—Cardiac dulness extends to middle sternum. Apex beat in fifth interspace in mid-clavicular line. Heart sounds are somewhat distant, second sound accentuated.

Pulse.—Arteries sclerosed, tension quite good.

Lungs.—Breathing regular. Auscultation and percussion negative. No râles are heard.

Eyes.—Pupils dilated, right slightly more than left. An external squint is noted in the right eye. The right pupil reacts sluggishly to light and accommodation; the left is more active.

Abdomen.—Scaphoid. A small mass is felt in the left hypochondriac region, dull on percussion.

Liver and Spleen.—Not enlarged.

Extremities.—Loss of knee-jerk; no ankle clonus; no Babinski sign.

Sensation.—Not impaired. On the lower part of the lumbar region, to the left of the spine, there is a large cauliflower-like growth which exudes sanious fluid. Over the sacrum there is a large area which is œdematous, reddened, and fluctuating. The glands of the groin on both sides are greatly enlarged.

Examined November 25, 1902. Has a slight ptosis of the right eye; has paresis also of internal rectus of right eye. Double vision is present in conjugate deviation to the left.

There is no facial inequality. Patient retracts the angles of the mouth equally well, and protrudes the tongue in the median line.

The right arm is paretic and ataxic. The left arm presents slight impairment of power, with very slight ataxia; paresis and ataxia are very much more marked in the right arm than in the left.

The legs present a paraplegia. Patient is unable to flex or extend the legs at the thighs; cannot flex them at the knees; has very feeble power of extension at the knees. Flexion and extension of the left foot are feebly preserved; these movements are absolutely lost in the right foot. Patient also has some preservation of flexion and extension of toes in left foot; these movements are also preserved in right foot, but to a very feeble extent.

Elbow-jerks and bicipital reflexes are lost. The knee-jerks are lost. There is no ankle clonus. Feeble extension of all the toes ensues upon plantar irritation of the left foot. There is no plantar reflex obtainable upon the right side.

November 27, 1902.—Feeble movement of flexion of toes is to-day elicited upon irritation of the inner side of the left foot. An area of tactile hypæsthesia exists over the dorsal aspect of right hand and fingers. There is also hypæsthesia to temperature and pain sense over the right arm, beginning near the axilla, and becoming somewhat more pronounced in fore-arm. In the right hand the temperature sense is lost completely. Deep impressions are recognized all over the hand; superficial impressions are not recognized. An area of hypæsthesia also exists on the dorsal aspect of the left foot, more especially over the distal portions of the outer two metatarsal bones.

There are no sensory losses in the left arm. Touch, temperature, and pain are well preserved. There are no sensory losses over the trunk or over the head. Tactile, pain, and temperature senses are well preserved in both thighs. An ill-defined hypæsthesia to touch, pain, and temperature exists in the right leg upon the posterior and inner aspects, below the knee. No sensory changes are found in the left leg below the knee.

Examination of Urine, January 2, 1903.—Color, yellow; cloudy; specific gravity, 1008; reaction, acid.

The microscopical examination revealed hyaline casts and pus cells.

The chemical analysis revealed albumen; no sugar.

January 6, 1903. Patient has been growing weaker for several days past. There has also been a gradual failure of mental faculties. Has had two severe general convulsions, epileptiform in character, in close succession. Afterward was delirious for several hours. There was no vomiting. Has developed diarrhœa.

The pupils unequal as before. The heart's action is rapid and weak. Has taken his nourishment fairly well. Complains of no pain or other disturbing symptoms.

The patient gradually grew weaker and finally died on the next morning, January 7, 1903.

The patient's temperature from admission, on November 20, 1902, was normal

until A. M. of December 4th, when it rose to 101.4°, and fell back to normal on the 5th; continued so until the evening of the 11th, when it rose to 101.1°, then fell back to normal until December 31st, when it rose to 102.3°, and then continued between 100° and 102° until January 3, 1903, when it fell back to normal, and remained so until morning of death, when it reached 101.3°.

Autopsy.—Body of a markedly emaciated adult male. Rigor mortis fairly well marked. Over entire surface of chest and abdomen, the anterior surface of the upper arm, and outer surface of hips, there are a great number of small nodules, varying from .2 to 1 cm. in diameter, and extending above the surface of the surrounding skin.

In the left lumbar region there is situated a tumor irregularly circular in outline. Its greatest diameter is 3 cm., and having an elevation of 1.5 cm. above the surrounding skin. The periphery of this tumor is nodular, of a bluish color, and apparently composed of altered skin. In the centre there is an eroded area, black in color, and having a smooth surface.

The glands of the groin are markedly enlarged on both sides. On opening the abdomen, the subcutaneous tissues were found to be studded with small, rounded nodules, varying from .2 to .8 cm. in diameter. The omentum is studded with nodules, as is also the mesentery. The parietal peritoneum contains a number of nodules; the mediastinal tissues are also studded with small nodules. The left pleural cavity is obliterated posteriorly by old adhesions. The right pleural cavity also contains adhesions. The pericardium contains about 60 cc. of colored fluid. Several small nodules are seen on the surface of the right ventricle, and also at the base of the large vessels. The right auricle contains clotted blood. The right ventricle is empty. The left side of the heart is empty; its wall is soft; the muscles are pale. The valves show no changes.

The left lung contains in the lower lobe an abnormal amount of fluid, reddish in color. At one place there is a dark area, about 3 cm. in diameter. The cut surfaces reveal a number of small nodules. The right lung shows changes similar to the left. The cut surface likewise shows nodules.

The left supra-renal body reveals numerous nodules in the peritoneal covering, but none in the parenchyma. The left kidney is large and studded with nodules. The ureters show a yellowish fluid resembling pus. The cut surface of the kidney is pale, the cortex is swollen, and a number of whitish nodules are noted. A number of grayish-yellow nodules extending from the surface penetrate to a depth of 1 or 2 cm. In the upper part of the kidney is a cavity 2 cm. in diameter. It contains yellowish fluid.

The right supra-renal body reveals conditions similar to the left. The right kidney presents a surface studded with nodules; the cut surface closely resembles that of the left.

The wall of the bladder is thickened, and shows inflammation of the mucosa.

The pelvis contains a quantity of fluid resembling pus.

The liver shows some evidences of cirrhosis. The cut surface is reddish and presents small grayish structures.

The retro-peritoneal glands are greatly enlarged, forming nodules, averaging about 4 cm. in diameter.

The surface of the pancreas is covered with nodules.

The brain is œdematous. Exudation of blood or hemorrhage has taken place at the base. The meninges show frequent small, grayish nodules. These are noted

Sarcomatous deposit on left postero-lateral aspect of cord.

at the base, but also in other situations, for instance, over the frontal lobes. Quite a large nodule, 2 cm. long and 1 cm. wide, is also observed on the internal surface of the dura over the left frontal lobe.

The spinal cord and its membranes seem large. In the lumbar portion a few gray nodules are seen.

Microscopical Examination.—The microscopical examination reveals numerous round-celled sarcomata, imbedded in the pia mater, both of the spinal cord and brain. In the spinal cord they are especially numerous in the lumbar region, though here and there nodules, small in size, are seen in the dorsal and cervical regions, and in one instance on the medulla. A somewhat larger mass than usual, oblong in shape, is found on the left postero-lateral aspect of the cord, in the lumbar region. The sarcomatous deposit infiltrates the cord substance itself in but a slight degree. Here and there, however, some invasion of the periphery of the cord is seen, as is shown in the accompanying illustration.

GUMMA OF THE IRIS AND CILIARY BODY WITH HISTOLOGICAL STUDY OF THE ENUCLEATED EYEBALL.

By G. E. DE SCHWEINITZ, A.M., M.D.,

PROFESSOR OF OPHTHALMOLOGY IN THE UNIVERSITY OF PENNSYLVANIA; OPHTHALMIC SURGEON TO THE PHILADELPHIA HOSPITAL.

The opportunity of enucleating an eye affected with gumma of the iris occurs but seldom, therefore the following record is contributed to the literature of this subject.[1]

Rose C., a married woman, aged 48, born in Pennsylvania, was admitted to the venereal ward of the Philadelphia Hospital on January 15, 1898.

History.—The patient's family history is unimportant. She has given birth to four healthy children, and has had one miscarriage. There is a history of several attacks of influenza during the last five or six years, with a good recovery in each instance. According to her statement, fifteen months before her admission to the hospital, she contracted syphilis from her husband, but there is good reason to believe that she not infrequently exposed herself to contagion from other sources, and therefore the exact date of inoculation is uncertain. Three weeks after the infection the earliest secondary lesions appeared upon the chest and limbs, and she suffered from fever, neuralgia, and sore throat.

Examination.—The patient was a medium-sized woman, who was liberally spotted with a diffuse, polymorphous, symmetrical, slightly copper-colored papular syphilide. There was moderate sore throat, with slight eruption on the mucous membrane, and a purulent vaginal discharge. The patient had a marked mitral systolic murmur and suffered much from cardiac asthma; urinary examination was negative.

One month after her admission to the hospital, iritis of the ordinary plastic type began in the right eye. After six weeks of treatment, which consisted of ascending doses of protiodide of mercury followed by iodide of potassium, the local manifestations of syphilis and the iritis disappeared, and the patient was dismissed from the wards.

On May 5, 1898, she was re-admitted to the hospital, this time to the ophthalmic wards, with the hope of finding relief from an inflammatory condition of the left eye which had existed for three weeks.

Examination revealed marked iritis, the pupillary margin of the iris being completely attached to the capsule of the lens, and the iris itself much discolored and thickened. In the lower portion of the anterior chamber there was a whitish, flocculent mass, while the upper and inner quadrant of the iris was occupied by a yellowish-white growth extending from the adherent pupillary edge to the ciliary

[1] A brief report of this case was made to the Ophthalmic Section of the College of Physicians, *November 21*, 1899. See Ophthalmic Record, ix., 1900, p. 39.

FIG. 1.

Gumma of iris and ciliary body.

border, beyond which the sclera was elevated as by a mass pushing it upward, bluish and discolored and bordered by a broken pigment line (Fig. 1). Vision amounted to faint light perception, the T was + 1, and the pain intolerable. The right eye, with the exception of the traces of the former iritis, exhibited no abnormalities, and vision, without correction, was 20/40.

The patient was given protiodide of mercury, later mercurial inunctions, and still later ascending doses of iodide of potassium. Of the last remedy she took for a long period between 100 and 200 grains a day, according to the condition of the stomach. Under the influence of this medication the broken-down gummatous material in the bottom of the anterior chamber disappeared, there was slight reduction in the flush of the eye, but there was no return of vision; in fact, toward the close of the treatment it is doubtful if the primary light perception still persisted. Through all this time the pain was considerable, often occurring in sharp exacerbations of typical glaucomatous type. Having faithfully tried this medication until June 27th, or, in other words, for seven weeks, without amelioration, enucleation was suggested to the patient, to which she readily consented. This operation was performed without accident; there was kind healing of the stump and entire disappearance of the pain. The woman was discharged on August 15, 1898, in apparently perfectly good condition.

The enucleated eyeball was hardened in formalin, 5 per cent., divided in equatorial section, one-half being reserved for macroscopic and the other for microscopic examination.

Macroscopic Examination.—The growth, which contains a small cavity, is seen to spring from and occupy the anterior end of the ciliary body. It includes and substitutes the iris as far as its pupillary margin, and entirely occludes the angle of the anterior chamber. Beneath, it is attached to the lens, which it invades and partially infiltrates. The opposite half of the iris is thickened and attached to the lens by its pupillary edge; its base is pushed against and adherent to the cornea. Behind the lens is the remains of the shrunken vitreous. The retina and choroid are in their normal positions.

Microscopic Examination.—Cornea.—The episcleral tissues are thickened, œdematous, and infiltrated with small round cells which are massed around the dilated vessels. These cells extend inward between Bowman's membrane and the corneal epithelium, and form a pannus tissue. On the upper part of the cornea there is a membrane formation which separates the epithelium from the pannus structure. Corneal corpuscles stain poorly, especially in the posterior layers.

Iris.—In the lower half of the iris, that is, in that portion not involved by the growth (and not shown in the drawing), it is not possible to trace the anterior endothelium, while the continuation of the uveal tract proper, that is the stroma zone, including the anterior boundary layer, the vascular stroma layer, and the posterior boundary layer, are densely infiltrated with small mononuclear round cells, which are grouped especially along the vascular channels and in the anterior layers. The anterior surface is covered with a layer of organizing cells in which some new-formed vessels are evident. In the stroma proper the vessels are thickened and the cells of the intima proliferated, so that some of them are entirely obliterated. At the pupillary margin the iris is thickened, intensely infiltrated with round cells and pigment grains and molecular detritus, where it is attached in a total synechia to the capsule of the lens. In contrast to the thickening of the pupillary margin of the iris, its periphery is atrophic, but an adhesive

inflammation at this position has fastened the iris to the posterior surface of the cornea and completely obliterated the filtration angle. The ciliary muscle is atrophic, the muscular tissue being replaced by connective tissue.

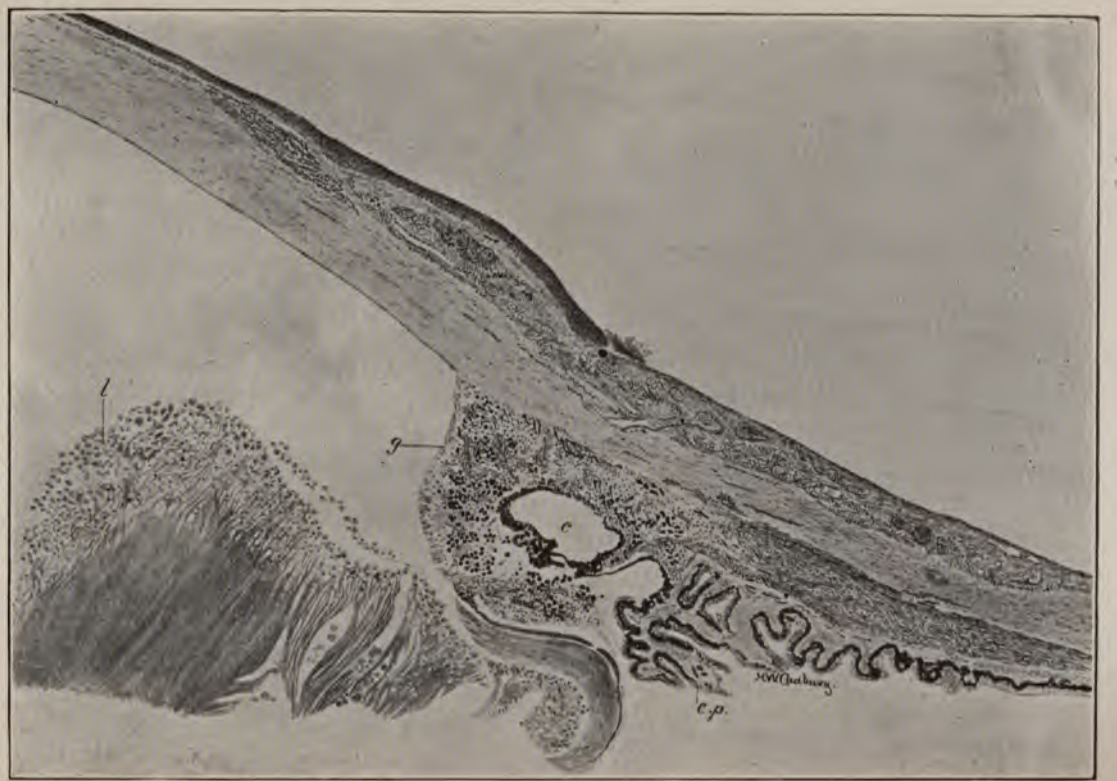
The upper half of the iris, or that portion occupied by the gumma, has almost been destroyed by the severity of the inflammation. The stroma is replaced by a large mass of small, mononuclear round cells in which the nuclei take up almost the entire cell. The cells all stain well, and there is no evidence of cellular degeneration at any point. Imbedded in the mass of round cells are the remains of the pigmented stroma cells (Fig. 2, *g.*). The posterior pigment layer is greatly proliferated. From the point of attachment to the cornea, well in front of Schlemm's canal, the iris bends backward and apparently doubles on itself, so that its anterior surface comes to lie in contact with the capsule of the lens. As it bends backward, a long process of proliferating pigment cells from the first ridge of the ciliary body extends forward to meet it, and forms with the posterior surface of the iris a wide cyst-like space, lined with a thick pigment layer (Fig. 2, *c.*). The iris is here filled with masses of large, round, densely pigmented cells. It shows very few blood-vessels, and these are fine capillaries, evidently newly formed and surrounded by mantles of round cells. The iris is adherent to the lens capsule, which is ruptured in the pupillary space and curls outward. The lens is cataractous and bulges forward into the anterior chamber. Its fibres are swollen and granular, and are infiltrated with cells from the iris (Fig. 2, *l.*). Most of the cells have likewise become œdematous, and swollen to large size, so that the nucleus occupies but a small part of the cell, and stains poorly. The anterior chamber is partly filled with coagulated fibrin.

The anterior portion of the ciliary body is destroyed by a round-celled infiltration similar to that which has been described in connection with the iris, with which, indeed, it is continuous. In other words, the process has destroyed or substituted the tissue of the iris and the anterior portion of the ciliary body. (Fig. 2, *g.*). The ciliary processes are converted into connective-tissue cords, which have undergone hyaline degeneration. (Fig. 2, *c.p.*). The blood-vessels are entirely obliterated. The angle of the anterior chamber at this position is obliterated by firm adhesion between the infiltrated iris and the cornea, and Descemet's membrane is destroyed in this position. Anteriorly the vitreous shows lines of infiltrated cells extending backward, but the process is not an extensive one. A coagulated exudate separates the retina from the choroid. Its inner portion is œdematous, and Müller's fibers are prominent. The vessels are hyperæmic, and are surrounded by considerable round-celled infiltrate. Many of them contain polymorphonuclear cells. The nerve-head contains a moderate cup, but its fibres are not at all or only slightly degenerated.

Fuchs,[1] in 1884, writing on this, or more accurately, on an analogous subject, syphilitic iritis, remarks: "Up to the present time only a few cases of syphilitic iritis have been accurately examined from the anatomical standpoint, chiefly because the difficulty of obtaining material. For the most part one is forced to examine pieces of iris which have been excised by an iridectomy, as, for example, Graefe and Colberg

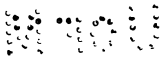
[1] Archiv f. Ophthal., Bd. xxx, Abth. 2, 1884, p. 1.

FIG. 2.



Gumma of iris and ciliary body.

g. Main portion of lesion substituting iris and involving anterior portion of ciliary body. *c*. Cyst-like cavity, bordered with pigment. *l*. Remains of crystalline lens infiltrated with swollen cells. *c.p.* Atrophied ciliary processes which show hyaline change.



and Michel have done." In this paper he reviews the literature available at that time. Since then a number of papers on gumma of the iris have appeared. Among the most importaut of these, references may be made to those of Brixa[1], De Lieto Vollaro[2], Busse[3], Ostwalt[4], Terson[5], Morton and Parsons[6], and Panas[7]. These articles contain, in addition to individual reports, a more or less thorough analysis of the existing literature.

In general terms, gummata of this region may be divided, as both Terson and Vollaro have done, into the precocious and tardy manifestation of the affection. It is, however, evident from Ostwalt's researches that very frequently specific lesions in the iris and ciliary body have been classified as gummas, when really they were not strictly of this nature, and he insists that a sharp distinction should be made between the syphilitic papule and the gumma, the former belonging to the secondary stage of syphilis, and appearing most frequently in association with an iritis, while the latter develops in the tertiary period, and at the time he wrote, namely, in 1896, with this distinction strictly in view, he was willing to classify only four of the examined cases as true gummas, adding one of his own observation to the list.

Vollaro, however, to quote from the abstract in Nagel's *Jahresbericht*, describes, both clinically and anatomically, seven cases from the clinic of Vincentiis, and refers to thirty other cases in the literature, five of which have been histologically examined. He makes evident the noteworthy fact of the early development of gumma in some cases, inasmuch as in six instances, at the expiration of six to fifteen months after the primary infection, the gumma appeared. In one case five years elapsed between infection and the gummatous manifestation. The course of the syphilis was unusually severe, and at the time of the appearance of the gumma skin and gland symptoms were

[1] Ueber Gumma des Ciliarkörpers und Luetische Augenhintergrundserkrankungen. Archiv f. Ophthal., xlviii. 1, 1899, p. 123.

[2] Sulle gomme del corpo ciliare. Contribuzione in rapporto al terziarismo precoce. Annali di Ottalm., xxviii, p 613, u. xxix, 1899, p. 47. Abst. Nagel's Jahresbericht, vol. xxx, p. 627.

[3] Zur Casuistik der syphilitischen Tumoren des Ciliarkörpers. Deutschmann's Beiträge zur Augenheilk., Heft. 4, 1893, p. 16.

[4] Cas typique de gomme du corps ciliaire, aves remarques sur les tumeurs, syphilitiques de la partie antérieure du tractus uvéal, en général. Revue Générale d'Ophth., 1896, p. 97.

[5] Gommes Précoces du corps ciliaire. Archiv d'Ophth., 16, 1896, p. 455.

[6] Gumma of the Ciliary Body. Trans. of the Ophthalmolog. Soc. of the United Kingdom, xxii, 1902, p. 266.

[7] Des gommes du corps ciliare, particulièrement de celles précoces. Archives d'Opht., vol. xxii, 1902, p. 485.

present, and once periostitis. Six times iritis was evident, and once secondary glaucoma. The development of the gumma occupied from twenty to sixty days. In spite of anti-syphilitic treatment, all cases came to enucleation, six on account of pain, and the one of chronic duration on account of sympathetic irritation.

The clinical picture in tardy or precocious development of gumma, according to Vollaro, is not essentially different, although in the early cases the inflammatory symptoms are more florid, and ulceration, caseation, and perforation developed more rapidly. Almost always, according to his observations, there is a preceding plastic iritis. After its disappearance, or while it is still evident, there is a relapse of inflammatory symptoms, and soon after at one or other points of the ciliary region dark-red elevations appear in the sclera. These grow larger, become gelatinous, gray-white, then yellow, and covered with yellowish and black points. The neighboring cornea becomes hazy, vascularized, and deposits appear on the membrane of Descemet. The anterior chamber is filled with a yellowish mass, inflammatory symptoms increase, the elevation becomes greater, perforation takes place, followed by atrophy. Rarely the form of the ball is preserved, and some preservation of vision is maintained.

Anatomically, Vollaro found the new formation to be composed of a compact mass which proceded out from the ciliary body to the sclera, cornea, and iris, and pushed into the anterior chamber, involved the lens and the anterior layers of the choroid, retina, and ciliary body. The growth itself was composed of granulation tissue, which, in irregular masses, infiltrated the separate tissues. These foci were composed of areas of epithelioid cells, in the centre of which more and more necrosis was manifest, and the periphery of which was constituted of a zone of rich, round-celled infiltration. The central necrosis was partly a fatty degeneration and partly a coagulation necrosis. Gradually scar-tissue develops. The posterior half of the iris did not exhibit very marked pathological alterations. In the vitreous, which was shrunken, fibrilla had formed, and its cells were vacuolated.

Terson, fully recognizing that syphilitic new formations, without a tendency to caseation, can appear in the ciliary body, and referring to the iritic condylomas which have been known and described for a long time, and which are almost always papules, to use Desmarres' words, and very rarely gummas, points out that the cases of precocious

gumma which he describes are of a different sort. Here the tendency is to caseation and rapid perforation, in spite of most active treatment, followed by atrophy of the eyeball, the lesion appearing in two or three months after the primary infection. He is impressed with the fact that usually the subjects have been comparatively young, that is, under thirty years of age, only two cases that he quotes, those described by Von Hippel and Gallenga, having past their fortieth year. While the tardy gumma of the ciliary body appears at the fourth or fifth year of syphilis, and often much later, these precocious manifestations develop themselves before the third year; for example, Von Hippel's patient was affected after two years, Panas's at the tenth month, Gallenga's at a year and a half, while in his cases the appearances were, respectively, after two and one-half months, after six months, and after two years. Like Vollaro, Terson has not noted any special difference in the symptomatology of precocious and tardy gumma. It appears almost always in the course of an intense iritis, with exclusion of the pupil, reduction of tension, violent pain, obliteration of vision and the rapid appearance of a retro-iridian process. The termination has been various—sometimes atrophy of the globe, in other cases enucleation was necessitated, but in his six cases a certain degree of vision was retained, in spite of scleral perforation.

Whether the case reported in the present paper should be classified as a true gumma of the iris and ciliary body is, in some respects, difficult to say. If one permits the time of the manifestation to influence the classification, then it did not appear in the gummatous stage of syphilis, having presented itself certainly not later than the eighteenth or twentieth month after primary infection. As we have already seen, however, this of itself is not sufficient to exclude a manifestation of this kind from the gummatous class. The intensity of the process, its utter failure to be influenced by the most vigorous anti-syphilitic treatment, the obliteration of vision, and the tendency to pass upward and involve the sclera, are symptoms associated with true gumma. Perforation, however, did not occur. Whether it would have occurred had the eye not been enucleated of course it is impossible to say. Histologically the process is not quite analogous to that which Vollaro has described in the cases of gumma which he examined, that is to say, there is a distinct absence of caseation and necrosis of the cells, if one excludes the cystic formation present in the centre of the new

formation, and yet, taking into consideration the clinical, and, to a certain extent, the histological signs, it would seem that the process is more elaborate than that which would be expected from an ordinary syphilitic papule, that the title of the paper, therefore, is justified, and that we deal in this case with a true gumma of the upper portion of the iris and anterior half of the ciliary body, tending to involve and perforate the sclera, which it had already elevated into a dark-red and partially pigmented ectasia.

OPERATION FOR ULCER OF THE STOMACH.

By J. CHALMERS DA COSTA, M.D.

The case occurred in the Surgical Wards of the Philadelphia Hospital. The patient's name was David Boulding. He was a colored man, forty-one years of age.

A note taken on May 5, 1902, says that for a week before admission he had been much nauseated, and had vomited a number of times, most of the spells of vomiting having been immediately after eating, and the vomit on several occasions having been stained with blood. Examination revealed the fact that there was marked epigastric tenderness, and pain limited to the upper portion of the abdomen. The man denied the existence of alcoholism or syphilis, and there was no history of tuberculosis or cancer in the family. During the past four years he had had three similar attacks. In each attack, when rectal feeding was instituted, he rapidly improved, and soon became apparently well. He is a healthy-looking, well-nourished man, with healthy lungs and heart.

The day after admission feeding by the mouth was absolutely suspended, and the patient was nourished exclusively on nutrient enemata. During the day he vomited at short intervals, and had a great deal of retching, and the material vomited was dark-brown in color and stained with blood. During the night he vomited about eight ounces of this bloody material.

Three days after admission there was a distinct improvement in his condition. The intervals between the attacks of vomiting became longer, the attacks were less severe, the vomited material consisted chiefly of mucus, with very little blood, and the pain was much diminished. In the epigastric region a small mass was detected by sight and by touch. This mass bulged somewhat on vomiting and on coughing but was dull on percussson, and was apparently a fat-hernia of the epigastric region.

May 10.—He was given a little peptonized milk at intervals during the day and night. It caused neither vomiting nor pain, and the enemas were stopped.

May 12.—Because of an aggravation of the condition, mouth feeding was again abandoned.

May 13.—The pain in the upper abdomen was very severe. The patient vomited small quantities of bloody matter frequently and was obviously weak. At this time an operation was advised, but was not at once accepted by the patient.

May 15.—The man was weaker, and palpation disclosed an extremely tender, circumscribed mass, midway between the ensiform cartilage and the umbilicus. On this day there was great pain, tenderness, and marked rigidity of the upper abdominal wall, and it seemed probable that a perforation was taking place. The patient consented to operation.

Incision of the abdominal wall showed that the mass in the epigastrium, which could be palpated, was a fat-hernia. It was drawing a part of the stomach downward, and would have eventuated in a hernia of the stomach. When the abdominal cavity was opened, an ulcer was found on the lesser curvature of the stomach, partly in the anterior wall and partly in the region of attachment of the gastro-hepatic omentum, and was nearer to the pyloric than to the cardiac end. There

was great induration; and in one place, over the centre of the indurated area, a large piece of omentum was attached. Right by the margin of this attached piece of omentum the floor was very much thinned, and almost ready to rupture. The ulcer was excised, several vessels tied, the mucus membrane was closed by a continuous suture of silk and two layers of silk. Halsted inversion stitches were employed to obtain a secure suture-line. Gauze drainage was carried down to the suture-line; the point of emergence of the fat-hernia was carefully sutured with chromic gut, and the wound in the skin was closed, except where the drain emerged.

The patient recovered from the operation quickly. He vomited a few ounces of bloody fluid, and complained of much pain during the night. Toward morning he vomited an ounce of pure blood. During the 16th and the 17th, he had frequent eructations of sour-smelling gas, and vomited and retched as he had before the operation; although, toward the evening of the 17th he vomited about six ounces of greenish fluid, which was free from blood. The abdomen was somewhat distended. During the 18th the condition was about the same. The fluid vomited was greenish, and was brought up without effort. It was necessary to give morphia to control the vomiting, all other treatment having failed; and the morphia was of great benefit. The patient was given purgative enemata to lessen the distension. Rectal feeding had been used at intervals since the operation, and stimulants had been administered in the same manner. On the 19th the patient's condition was desperate, and he himself became hopeless. Attempts to administer small amounts of food by the mouth failed, as they were at once rejected. The patient was very weak, with a rapid pulse and a distended abdomen.

On the 20th small quantities of champagne were given, with the most beneficial results; the vomiting ceased, and the distention lessened. The gauze drain was removed from the abdomen on the 20th, and on the 21st the patient's condition was noticed to be much improved. Some liquid beef peptonoids were given and retained—the first food successfully given by the mouth for fifteen days. Rectal feeding was still continued. Vomiting had ceased, and nausea was only trivial. On the 25th the patient was receiving small amounts of food by the mouth. Once during the night about six ounces of greenish fluid were vomited. On the 30th the patient's condition was so much improved that predigested nourishment by the mouth was increased.

On the 30th the improvement was decided, and nutritive enemata were abandoned. On June 5th soft house-diet was administered, and from this date there was no further vomiting. It is interesting to note that attacks of vomiting occurred during nineteen days after the operation. On June 10th the patient had apparently completely recovered.

The following is the report of the pathologist, Prof. Coplin.

The specimen in alcohol, as delivered at the laboratory, weighs 6.85 gms., and consists of a fairly regular, slightly oval, discoidal piece of tissue, measuring 3.5 cm. by 2.5 cm., and from 1 to 1.5 cm. in thickness; it presents for description two surfaces and one margin.

One surface is partly covered by what appears to be mucosa; this is the larger of the two surfaces. Its edge is irregular, and tends to bend around the free margin of the specimen. The surface is beset by irregular depressions, usually linear,

approximately 1 mm. in depth, showing a tendency to radiate from a slightly eccentric excavation to be described later.

Between the depressions just described are miniature rugæ possessing the same radiating tendency as the depressions. At other points, instead of rugæ, the surface is mammilliform or mammillated, the miniature mammillæ not exceeeding 1 to 2 mm. in height or diameter. This surface is rather soft and grayish in color. Near one end of this aspect of the specimen is a crater-like depression, apparently an ulcer, 0.7 cm. in depth. In contour the ulcer is oval, its long axis 1.4 cm., corresponding to the long axis of the specimen; transversely it measures 0.7 cm. The margin is depressed or inverted—folding over and passing downward toward the floor of the ulcer; it is not perfect, having been torn or cut at one edge. At one point the mucosa projects over the side and floor of the depression—a darker fragment of the mucosa 0.5 by 0.25 cm. being suspended by a pedicle 2 or 3 mm. in diameter.

The floor is occupied by grayish-brown detritis that appears to be loosely attached, but it is thought best not to remove it. The sides (except the overhanging mucosa, as already mentioned) are curved outward and then inward, uniting with the necrotic material already described as occupying the floor.

The opposite surface is harder, smoother, and flatter than that just described. At a number of points it is finely granular, and toward one margin is a slightly purplish spot, 0.25 cm. in diameter.

The margin of the specimen, at a suitably selected part, shows three distinct strata. (1) A mucosa, 2 to 3 mm. in thickness. (2) A grayish-white, dense, elastic layer that forms the major part of the thickness of specimen and measures, at one point, nearly 1 cm. While mostly grayish-white it shows oval or irregularly oval semi-translucent and darker areas, 1 to 2 or 3 mm. in diameter, that probably are sectional views of the residual smooth muscle elements, separated and grouped by intercallated bands of fibrous tissue. There are at points suggestions that these might be divided into layers, but the appearances of stratification are not convincing. (3) A very thin, scarcely measurable layer, not over 1 mm. in thickness, apparently corresponding to the serosa.

The specimen has been greatly distorted by the fixation contraction of strata (2) and (3), and it is not possible satisfactorily to identify a submucosa or subserosa, although, of course, both were present. Without sectioning the mass one cannot say how near to the serosa the floor of the ulcer extends, but by measurements at this point it seems safe to conclude that the residual tissue in the contracted specimen does not exceed 0.25 cm. in thickness.

The case presents several features of considerable interest: In the first place, the fat-hernia of the epigastric region, which would have been the forerunner of a hernia of the stomach itself. It is probably thus that true gastric herniæ are usually formed.

In performing the operation upon the stomach, it was deemed wise to excise the ulcer; because the great and widespread induration and the situation upon the lesser curvature rendered successful inversion of the whole area difficult or impossible. Furthermore, had such inversion been effected, there would have been turned into the cavity of the

stomach a large mass of ill-nourished tissue that would have been extremely liable to undergo necrosis. In the acute perforating ulcers of the stomach there is usually little induration, and excision is not required; but in perforation in a chronic ulcer excision is usually demanded.

It is interesting, also, to note that the ulcer had all but perforated, and that a piece of omentum had applied itself as a barrier at the point at which perforation was about to occur. This mysterious action of the omentum, which has been recognized by all clinical surgeons, is extremely valuable, but is brought about we know not how.

Drainage was carried down upon the suture line, and I believe that this precaution should always be taken. It is quite true that many cases of operation upon the stomach have recovered when drainage was not used, but it is equally true that now and then a suture line leaks. If drainage has not been used, a leaking suture line will, in all probability, kill the patient; but if drainage has been used, leaking will not of necessity involve a fatal termination. There are, of course, objections to drainage, and in this case the man's condition was improved when the gauze was removed, but I think the balance of safety is on the side of drainage.

A FATAL CASE OF POLYARTHRITIS COMPLICATED BY CHOREIFORM SYMPTOMS AND VEGETATIVE ENDOCARDITIS.

By AUGUSTUS A. ESHNER, M.D.

PROFESSOR OF CLINICAL MEDICINE IN THE PHILADELPHIA POLYCLINIC; PHYSICIAN TO THE PHILADELPHIA HOSPITAL; ASSISTANT PHYSICIAN TO THE ORTHOPÆDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES.

A young man, aged seventeen years, was admitted to the medical wards of the Philadelphia Hospital on May 18, 1902, with a history of having suffered for two weeks with pain in various joints, beginning in one knee, then invading the other knee, and next extending to the ankles, and finally appearing in the elbows. He had slight elevation of temperature—not exceeding 100.2°—and corresponding acceleration of pulse and respiration. The only abnormality noted on physical examination was a "hemic heart murmur" and a bruit in the vessels of the neck. The urine contained neither albumin nor sugar. The family history was unimportant except for the statement that a sister had been subject to rheumatism, and had died in an attack of chorea, and the only significant point in the personal history was a previous attack of "rheumatism." With rest in bed and administration of salicylates the youth improved, and at the end of the week he was so far recovered as to request his dismissal, and he was discharged on May 26. He was, however, readmitted on June 15, again complaining of pain in various joints—knees, ankles, and wrists—that had set in soon after his return home, and had been present since. There were also some stiffness and swelling, and the patient was sweating freely. He had had a fainting attack the night before entering the hospital. Apart from harsh breathing and a few dry râles no physical abnormality was detected. The patient was noticed to be nervous and emotional, and on the 21st he began to sob and cry for long periods at a time. His restlessness gradually increased and he became unable to sleep. He tossed about violently, and at times attempted to get out of bed. He cried out loudly in an inarticulate manner. The hands and feet were in almost continuous choreiform movement. The knee-jerks at this time were found enfeebled, and the plantar reflex absent on the left and present on the right. The patient gradually grew weaker, and finally died from exhaustion early on the morning of July 4th. The temperature fluctuated in the neighborhood of 100° for the first four days, becoming normal on the morning of the 5th. It rose slightly, however, on the following day, and it subsequently pursued an irregular course, reaching its highest, 102.6°, on the tenth day. Pulse and respiration pursued a corresponding course, but toward the close became greatly exaggerated, reaching 129 and 44 respectively. The urine yielded a slight reaction for albumin.

On postmortem examination rigor mortis was present. The pupils were equal. Both lower extremities were deflected to the left, and there was fairly well-marked talipes equinus varus. The joints of the lower extremities were not obviously

enlarged, although the right wrist appeared fuller than the left. The muscles, on section, were exceedingly dark. The peritoneum was smooth and glistening, and the abdominal cavity contained no excess of fluid. The mesenteric glands were slightly enlarged and soft, and some were hemorrhagic. The right lung was congested and bound down by old adhesions. The heart was not increased in size. Its epicardial surface was smooth, the musculature dark brown in color, with yellow mottling beneath the endocardium. The wall of the left ventricle was slightly thickened, but the cavities of the heart were normal. The tricuspid and pulmonary valves also were normal. The mitral valve was thickened, and along the free edge of its leaflets dark-gray, translucent vegetations were visible. The aortic leaflets exhibited somewhat similar bunches of vegetations immediately below the corpora arantii. Some of the vegetations were about the size of a pea. Otherwise the valves were delicate and smooth. The spleen was slightly enlarged and soft. Its surface was smooth and dark violet in color. On section it presented a purplish-red appearance. Its substance was soft, but not friable. The Malpighian bodies and the visible trabeculæ were not increased. The liver was fairly large, smooth, and soft. Its surface was red, with yellow mottling. Its cut surface was smooth, and its lobules were fairly well marked. The periphery was yellowish, the centre somewhat congested. The capsules of the kidneys stripped readily, leaving a smooth surface. On section the cortex appeared increased, and the entire surface was cloudy and gray. The striæ were fairly well marked, and the glomeruli generally were injected. The common bile-duct and the gall-bladder were normal, although the former was obstructed by an enlarged gland near the duodenum. The stomach was congested. The œsophagus and the intestines were normal, as were also the pancreas, the adrenals, the bladder, and the prostate. The right wrist was opened and the cartilaginous extremity of the radius and the ulna exposed. The joint contained no excess of fluid, and the cartilaginous surface appeared normal. The synovial membrane was, perhaps, a little hyperplastic, though delicate. The related tendons and sheaths were normal. The brain and cord were put aside for examination, but in consequence of some defect in technic factitious cavities developed. Careful macroscopic and microscopic examination by Dr. D. J. McCarthy failed to disclose any abnormality.

This case is interesting by reason of the association of polyarthritis, choreiform symptoms, and endocarditis. I prefer to speak of "polyarthritis" rather than "rheumatism," and of "choreiform symptoms" rather than "chorea." Rheumatism represents but one form—I am inclined to believe a specific form—of polyarthritis, but the differential criteria of the several varieties are as yet too uncertain to permit of dogmatism in this connection. It is all but certain that rheumatism is an infectious disease, and it seems probable that it is dependent upon one of the several cocci that have been isolated from the affected joints and endocardium, and some of which it has been possible to cultivate upon artificial media, and by injection of cultures produce arthritis and endocarditis in lower animals, the same organisms again being obtained. Chorea likewise is looked upon as an infec-

tious disease, but nothing is known definitely of the exciting cause. The frequent association of articular symptoms and valvular lesions of the heart with chorea is strongly suggestive of a close etiologic relation between this disease and polyarthritis—possibly rheumatism, but whether the exciting agent is the same in both instances obviously cannot be determined in the present state of knowledge. It is to be regretted that no bacteriologic investigation was undertaken in the case here reported, as it might have been possible by this means to have determined the etiologic factors, and to have shed some light upon the general subjects of chorea and rheumatism, and the relations, if any, that exist between these two affections. We have learned that various cocci may each give rise to a number of distinct lesions, and it may eventually be found that rheumatism and chorea merely represent different localizations of the same infection. This apparently is the view held by Leonard Guthrie (*British Medical Journal*, May 18, 1901), who suggests that chorea is not a disease in itself, but merely a manifestation of rheumatism. A concrete instance of this relationship is reported by Westphal, Wassermann, and Malkoff (*Berliner klinische Wochenschrift*, 1899, No. 29), who record a case of acute articular rheumatism, followed by chorea and complicated by endocarditis, in which they succeeded in isolating from the blood, the brain, and the endocardial vegetations a streptococcus capable of exciting polyarthritis in lower animals.

M. Reichardt (*Deutsches Archiv für klinische Medicin*, 72. B., 5. u. 6. H., p. 504) has recently recorded the results of postmortem examination in two fatal cases of chorea, one occurring in the sequence of an attack of rheumatism. In both vegetative endocarditis was present, staphylococcus aureus being isolated from the heart's blood in one case and cocci in masses and chains from the vegetations in the other. In both also inflammation, hemorrhage, and degeneration of nerve-fibres in the central nervous system were found on histologic examination.

REPORT OF A CASE OF SPLENIC PSEUDOLEUKÆMIA.

By THOMAS G. ASHTON, M.D.
(Pathological report by A. F. COCA, M.D.)

Although the pathological findings in the present case are of paramount interest, the clinical history is, nevertheless, not without importance.

The patient, Antonio De G., a native of Italy, aged twenty-eight years, entered my wards in the Philadelphia Hospital on February 10, 1902, complaining chiefly of pain in the left abdominal region, weakness, headache, and anorexia.

The family history has no bearing upon the case, his father and three brothers dying of some acute disease, each after a few days' illness, while his mother is still living, and well.

The patient had been in this country for five months, having previously lived in the northern part of Italy, where he pursued the occupation of a laborer, chiefly as a worker of the ground. Alcohol and tobacco had both been used to excess, but any venereal history was denied.

No history of a previous illness could be obtained, with the exception of an attack of chills and fever seven months preceding his admission to the hospital, and while the patient was still a resident of Italy. With this attack he was ill one week, the ague yielding readily to treatment, and though it was probably caused by a malarial infection, it was attended by urgent vomiting and pain over the splenic area, symptoms that were prominent features of his case while under our care.

Nevertheless he remained subjectively in perfectly good health until the middle of January of the present year, or about four weeks before first coming under our observation. At that time he was taken somewhat suddenly ill with general pains, fever, but no chilliness, and marked distress over the region of the spleen.

While in the hospital these symptoms persisted, and were superadded to by diarrhœa, intractable vomiting, anorexia, loss of weight, profound asthenia, and headache; while a mild cough with occasional blood-tinged expectoration had existed for about two months. The case pursued a febrile course throughout, the temperature being of an irregular type.

The physical examination showed a considerably emaciated adult male, with a muddy or slightly icteroid hue to the skin, and an anxious expression. The mucous membranes were pale; the tongue thin, coated, and extremely pale, and a herpetic eruption about the lips. The eyes were sunken, the conjunctivæ pale and slightly tinged with yellow, the pupils reacting normally.

Visible pulsation of the vessels of the neck was observed. The pulse was regular but rapid and compressible. The apex-beat of the heart, although not visible, was found upon palpation to occupy its normal position. At the apex a soft systolic murmur was heard, but was not transmitted, and a faint systolic murmur was also detected at the left base over the pulmonary area.

The lungs generally were hyperresonant except over the right upper lobe anteriorly, where the percussion note was less resonant, and where at the necropsy extensive pleural adhesions were found. Numerous subcrepitant and submucous râles were heard throughout, being especially numerous at the bases.

The abdomen was greatly enlarged, particularly on the left side, due in great part to a marked increase in the size of the spleen. The area of splenic dulness extended in the anterior axillary line from the sixth intercostal space to a point one inch below a line drawn transversely through the umbilicus. The anterior border passed forward as far as the umbilicus and above merged with hepatic dulness; it presented two distinct notchings. Palpation of the spleen caused much distress. Immediately below the costal border over the spleen a distinct blowing murmur could be heared.

Liver dulness in the right mammillary line began above, in the fifth interspace, and extended about two inches below the costal margin, the left lobe also showing some increase in size. Palpation of the liver caused no tenderness.

The superficial lymphatic glands were not enlarged, and there were no symptoms of involvement of the nervous system; neither at any time were there any hemorrhagic manifestations. Extreme tenderness of the sternum and tibiæ was present.

An ophthalmoscopic examination made by Dr. Oliver showed marked neuroretinitis in both eyes, and aggregates of leukocytes and areas of partially absorbed erythrocites, most pronounced in the region of the disks and maculæ. The retinal veins were engorged and the eye-grounds extremely pallid. Conjunctivæ blanched.

Examination of the urine gave a specific gravity of 1012; acid reaction; no sugar; a decided amount of albumin. Microscopically, coarse granular and granular epithelial casts were found.

Examination of the sputum for tubercle bacilli gave negative results.

The blood, examined first on February 12th, the second day after the patient's admission, showed the erythrocytes to number 1,610,000; the leukocytes, 5600, and the hæmoglobin to be 20 per cent. The erythrocytes were much deformed, and there were numerous microcytes and macrocytes. No nucleated cells were present. On February 15th the blood count was substantially the same; the hæmoglobin, however, had fallen to 16 per cent.

Differential estimation showed the leukocytes to be made up as follows: Polymorphonuclear, 58 per cent.; large mononuclear, 21 per cent.; small mononuclear, 14 per cent.; transitional forms, 3½ per cent.; eosinophiles, 3½ per cent.

The final blood count was made on February 24th, and showed a decided falling off in the number of erythrocytes, the number then being 1,100,000; leukocytes, 5800, and hæmoglobin, 16 per cent. The differential count of the leukocytes showed no material change from the previous examinations.

On February 26th the patient died.

Pathological Findings.—The autopsy of this case was performed by Professor Flexner, to whom the writer is indebted for the privilege of reporting it. His report is as follows:

Autopsy held five hours after death. Body of slightly built, emaciated male. Pigmented surface, sallow. No œdema. Pigmentation slightly more marked over pubes. Mucous membranes pale. Pupils equal and moderately dilated. At the outer side of the right foot is an old scar. Abdomen moderately distended. No

rigor mortis. Subcutaneous fat slight. Muscles pale; omentum free; small amount of fat; 60 c.c. of serum in abdominal cavity.

Liver.—Free from adhesions; 7 cm. below costal margin on right side; 6 cm on left; 9 cm. below ensiform cartilage. *Right lung* bound; left lung free; in each pleural cavity 200 c.c. of clear fluid. *Pericardium* contains 40 c.c. of clear, yellow serum; both layers smooth; visceral layer œdematous. *Heart* valves normal; heart flesh pale and friable. *Left lung* does not retract; is pale and generally emphysematous. *Right lung*, upper lobe posteriorly, shows congested area, triangular in shape, 2 x 3 cm., showing appearance of infarction. *Spleen* much enlarged, extending to line drawn transversely across abdomen at umbilicus. Weight, 2000 grammes, bound to diaphragm and abdominal wall. It is very firm; on section, it presents uniform brownish-red color. *Liver* is smooth, of rusty brown color; on section, uniform rusty brown color. *Pancreas* is firm and gray. *Kidneys*, capsule strips easily; mottled; cortex swollen; striæ pronounced; pyramids injected. *Adrenals* small, but apparently normal. *Stomach*, mucous membrane is soft; otherwise normal. *Small intestine*, mucous membrane pale. *Large intestine*, mucous membrane much congested. *Bone-marrow* (femur) is red, soft, lymphoid. *Larynx*, at conjunction of cricoid and thyroid on left side, is an area of necrosis, 2 x 2 cm., extending into the cartilage, which is dark and gangrenous. *Left tonsil* shows circumscribed swelling the size of a bean; surface congested. *Gastrohepatic lymph glands* swollen and of rusty brown color. Remaining lymph glands not prominent. On both sides of the dura mater are found petechial ecchymoses.

Pathological Diagnosis.—Grave anæmia; chronic splenic tumor; pigmented liver; gastrohepatic lymph glands; lymphoid bone-marrow; pulmonary emphysema and lobular hepatization; swelling of left tonsil; circumscribed necrosis of larynx; petechial ecchymoses on both sides of the dura mater.

Bacteriology.—Cultures from liver, spleen, and kidney remained sterile after a week's incubation.

Spleen.—The capsule is considerably thickened, but otherwise normal. There is a marked increase in the splenic pulp in the amount of connective tissue of both the white fibrous and elastic varieties. The Malpighian bodies are generally sclerosed, the process often being most marked in the immediate neighborhood of the bloodvessels. The walls of the latter are hyaline. Hyaline flakes are found in a few of the Malpighian bodies, collected in the longitudinal masses. The blood sinuses are congested, and contain many red cells, which stain poorly or not at all; also mononuclear eosinophiles, lymphoid cells, and large cells with multilobulate nuclei identical with those found in bone-marrow. Numbers of cells are found containing spherical hyaline acidophilic bodies, measuring from 1 to 10 μ in diameter. These bodies are also found in small collections, lying free in the spaces of the splenic reticulum. No normoblasts are found. A number of karyokinetic phases occur in cells of the splenic pulp. Many large cells with large single vesicular nuclei and prominent nucleolus are caught in the act of amœboid movement. The protoplasm of these cells is abundant, and, with hæmatoxylin, take a purplish tint.

The wall of one of the large splenic vessels is broken through in two places, and the lumen invaded by tissue continuous and identical with the splenic pulp. Immediately beneath the endothelium of this vessel is found a layer of lymphoid and red blood cells.

Bone-marrow.—Smears stained by Jenner's method show nothing abnormal.

Sections show complete absence of fat and increased numbers of the normal cellular elements, especially of mononuclear neutrophiles and eosinophiles, but no noticeable increase in the number of either normoblasts or lymphoid cells. No megaloblasts are found.

Lymph Glands (gastrohepatic).—The connective tissue in the glands is increased; no karyokinesis is observed. The hyaline acidophilic bodies described in the spleen are found in the same situations here. There is a marked dilatation of the efferent lymphatics, in which pigment-bearing cells are found. Two are *hæmolymph* glands with typical peripheral blood sinus, in which are found nucleated red cells, marrow cells, and the multilobulate cells of bone-marrow. Mast cells are absent. A coarsely granular golden-yellow to yellowish-brown iron-bearing pigment is found: 1. In large cells with oval, reniform, or rounded and usually eccentric nuclei, and possessing abundant protoplasm, lying unattached in the reticular spaces. The nucleus is frequently concealed by the abundance of pigment. 2. In the proper cells of the reticulum. 3. Lying free in the substance of the reticulum itself and in the reticular spaces.

Liver.—The hepatic cells are generally swollen, and show early fatty metamorphosis, this being most marked in the neighborhood of the hepatic vein. The capillaries are greatly dilated, and contain an abnormally large proportion of lymphocytes and a few mononuclear eosinophiles and neutrophiles. One multilobulate marrow cell is found. No nucleated red cells. Connective tissue not increased. Capsule normal. Numbers of mast cells are found in the connective tissue of the interlobular spaces. A pigment identical with that found in the lymph nodes is present: 1. In the hepatic cells. 2. In the endothelial cells of the capillaries. 3. In the connective tissue cells, endothelial cells, and spaces of the wall of the portal vein. In detached endothelioid and hepatic cells, and lying free in the lumen of the portal vein. 4. In the endothelial cells lining the hepatic vein; in detached endothelial and liver cells in the lumen of the hepatic vein. No iron-free pigment is present.

Kidney.—There is marked general congestion, which, however, spares the glomeruli. No hemorrhages are found. The glomeruli show no changes. The tubular epithelium is very granular, and many of the cells are disintegrating into the lumen of the tubules. Numerous pyknotic nuclei are seen. Capsule normal. Connective tissue not increased.

Tonsil.—Enlarged tonsil shows general sclerosis, especially in the neighborhood of the vessels. The follicles possess wide central areas of endothelioid cells. Crypts contain degenerated epithelium and polynuclear leukocytes. The vessels are congested.

Larynx.—Sections studied do not include cartilarge. There is a broad superficial area of necrosis not sharply defined. The surrounding tissue is infiltrated with neutrophiles and eosinophiles, the former predominating. In the necrotic tissue are found great numbers of bacteria, those near the surface being streptococci and staphylococci, bacilli, and a long, unbranched organism, staining irregularly by Gram's method, hæmatoxylin, Babe's safranin, and not stained by iodine, and of various lengths, corresponding to the leptothrix maxima buccalis of Müller. In the deeper portion of this tissue only the leptothrix is found.

Lung.—The triangular area resembling an infarct proved to be an area of pneumonic consolidation, with the usual characters.

The pathological examinations of the case marks it as a typical one of its kind. No new light can be thrown upon the etiology of the disease by the present study, and the main interest seems to be found in the accidents of the case, especially in the study of the hæmolymph glands and the comparison of the disease under consideration with that described by Banti under the name of "splenomegaly with cirrhosis," now known as "Banti's" disease.

There is at present great difference of opinion among anatomists and pathologists as to the function or functions of the hæmolymph glands, the latest contributions being from Ann Arbor, by Dr. Warthin, and from Italy, by Morandi and Sisto. The latter believe that nucleated red cells are never found in hæmolymph glands, and do not admit hæmoblastic functions to them under any circumstances; the glands of our case contain normoblasts in sufficient number to establish the existence of such a function, in view of the fact that the blood of the patient before death was entirely free from nucleated red corpuscles. The absence of mast cells is a noteworthy aberration from the normal.

The pathology of this case corresponds very closely with that of Banti's disease, one feature alone being absent—the cirrhosis of the liver. The sclerosis of the spleen seems to proceed much in the same manner as described by Banti under the name of "fibroadenie."

A study of the statistics of splenectomy in such cases as this makes it remarkable that this procedure is not resorted to more frequently for their treatment, this appearing to be the only certain method of cure.

The literature of the subjects considered in this report may be found as follows:

Splenic anemia: Literature collected by B. W. Sippy in *American Journal of the Medical Sciences*, vol. cxviii.

Banti's disease: Literature collected by Banti in *Ziegler's Beiträge*, vol. xxiv.

Hæmolymph glands: Literature collected by Aldred Scott Warthin, in *Journal of Medical Research*, May, 1902.

BRIEF REPORT UPON THE TREATMENT OF GONO-COCCAL CONJUNCTIVITIS.

By CHARLES A. OLIVER, A.M., M.D.,
OPHTHALMIC SURGEON TO THE HOSPITAL.

As the result of personal studies extending over several terms of service during five years' association with the hospital as one of its ophthalmic surgeons, the writer (who, with the exception of one month during that period of time, has never been without undoubted cases of this kind), has, through many trials of various plans of therapy, arrived at methods of treatment which in his hands have resulted in the loss of but three eyes: In these three cases the diseased condition was so advanced when first seen that the eyes were already practically useless.

Recognizing the metatrophicity of the type of germ life he has had to deal with; the intimate relationship between the local conditions and the general state; and knowing the relative powers of the various medicaments he has tested during the treatment of his cases, the writer has long since come to the conclusion that given an early case of gonococcal conjunctivitis, even in the most asthenic adult (the worst type of all), no fear as to fatal, or even indifferent ocular results, need be entertained if the following plan of treatment be judiciously observed in each individual case. Cared for in this way, this once most formidable disease may now be considered a comparatively non-dangerous germ type of ocular affection in spite of the present want of a specific treatment.

The chief requirements may be enumerated as follows:

(1.) Free and sufficiently often repeated, but gentle, cleansing of the surfaces of the infected tissues. Thorough testing of the different vaunted washes has brought the writer back to the use of careful and frequent flushing of the exposed parts with sterile water; this being best applied through a bulb syringe by a nurse who has learned to perform the technique with little or no damage to the delicate and inflamed structures.

(2.) Early obtainment and maintenance of full pupillary dilation, together with the removal of all undue iridic and ciliary body muscle action. These purposes can best be fulfilled by the use of atropin, or, when necessary, scopolamin supplemented by the employment of the former drug.

(3.) Lowering the vitality of the invading cell form into an involutional condition, with consequent lessening of ability to multiply. This may be in a measure accomplished by the constant use of iced compresses, care always being taken that the ice is applied only to those eyes which this form of therapy will not injure.

(4.) Maintenance of the vitality of the organ, particularly in cases in which there are trophic disturbances, as, for example, in corneal involvement. This result is best obtained by the topical application of heat in spite of the favorable influences of this agent upon the reproduction of germ life.

(5.) Destruction of the intruding floral germ cell material with as little disturbance as possible to the surrounding faunal cell forms of the involved parts. This is most easily and readily accomplished by the cauterizing effect of nascent nitric acid obtained by gently patting the exposed areas of the palpebral mucous structures with weak solutions of nitrate of silver, by which the germs are destroyed, and their nesting grounds of open serous cell cavities and lymph and vascular channels either closed or annihilated.

(6.) Local and general isolation, together with treatment of similar infections of other mucous surfaces or serous tracts, both by local and general medication, in order that the affected ocular tissues may heal the more readily. The first two of these conditions are best obtained by segregating the patient and covering the unaffected eye with a well ventilated protective shield to prevent its infection—or where both eyes are simultaneously affected, by keeping the dressings separated—thus lessening the chances of continual autoinfection. General medication must depend upon the indications presented. (For example, in the chronic blenorrhœa, such as gleet, the writer has accomplished the best results in the treatment of the ocular complications by combining the internal exhibition of such mucous stimulents as cubebs, copaiba, and oil of sandalwood with the local measures. Where there are acute forms of the infection in other mucous membranes, such as an active urethritis, he has resorted to those local measures which

seemed to be best adapted to the individual case. In general gonococcal infection he has made good use of general mucous tonics, while once or twice he has employed hypodermatically graded doses of formalin.)

(7.) Support and improvement of the general condition of the patient. This, which permits the system to exert its local defensive influences to their utmost, is best secured by the correct and copious ingestion of easily digested and assimilable food-stuffs, the graded internal use of plenty of sterile water, strict emunctorial hygiene, and the employment of organic correctives when needed.

(8.) Employment of constant skilled nursing as long as bacteriological study gives evidence of the presence of the offending germ. This is practically the most important of all the conditions necessary for successful treatment of this affection. The writer feels sure that the good results in his series of cases has been largely due to the constant and assiduous care of the trained nurses in this hospital who have by a large experience become skilled in the handling of such cases.

All these rules for medication and hygiene must be carefully regulated and graded in strict accordance not only with the coarse physical appearances of the disease, but also by daily, and even tri-daily, study of the quantity, the condition, and the apparent activity of the gonococci upon the desquamated cell forms.

Recurrences should be guarded against by a gradual cessation of treatment, particularly of the applications of cold, and no case can be considered free from the need of isolation and constant care of special nursing until after three or four days (or even more) have elapsed since the last appearance of the germ-life in the excretions of the affected parts. Theoretically, cure can be assured only when it has been proven that gonococcal infection has been completely eradicated from the system.

THE TREATMENT OF PURULENT OPHTHALMIA.

By HOWARD F. HANSELL, A.M., M.D.,

CLINICAL PROFESSOR OF OPHTHALMOLOGY, JEFFERSON MEDICAL COLLEGE; PROFESSOR OF DISEASES OF THE EYE, PHILADELPHIA POLYCLINIC; OPHTHALMOLOGIST TO THE PHILADELPHIA HOSPITAL.

---

1. *Preventive.*—The prophylaxis of ophthalmia neonatorum has been exploited for many years and so frequently that there remains nothing new to add, although corroborative testimony may have its value. The great worth of the Credé method of cleansing of the eyes of the newborn, and of rendering aseptic the birth canal for some days preceding confinement, has been conceded by all competent observers. The suggestion of the nitrate of silver prophylaxis was made before the study of the bacteria of the conjunctiva, and to-day, when the gonococcus of Neisser is as much dreaded by the ophthalmologist as the storm clouds by the mariner, the method is almost universally practised. Clinical records carefully kept have shown that the percentage of cases of ophthalmia neonatorum has been reduced many-fold, and as an important and practical sequence the number of blind, in asylums from this cause, greatly lessened. The prevention and early treatment of the disease is of vital consequence to the community. The authorities have recognized the necessity for government interference, and many of the states of the United States, after urgent and repeated appeals from the medical profession, headed by Dr. Lucien Howe, of Buffalo, N. Y., have adopted laws enforcing the immediate report of cases of incipient inflammation of the conjunctiva as cases of contagion, with a penalty attached for the non-observance of the law. This drastic measure has resulted in good. There have been no convictions, or very few, for breaking the law, but the attention of physicians and midwives has been forcibly called to the disease, its prevention and cure, and to the bad economics and greater criminality in filling asylums with children, blind from a preventable and curable disease.

Modifications of the silver nitrate method of prophylaxis, consisting of the use of other antiseptics than silver nitrate, because of the supposed damage to the cornea and conjunctiva from the silver, have been

proposed, but they are less reliable, equally harmful—silver nitrate solution in proper strength and skillfully applied is harmless—and possess no advantages. The claim that silver nitrate is injurious to the ocular tissues has not been substantiated. The source of the injury might with greater justice be laid at the door of the unskillful nurse or accoucheur. I have seen a case of traumatic kerato-conjunctivitis diagnosed as infectious conjunctivitis in which the conjunctival inflammation was secondary to a wound of the cornea produced by the finger-nail of the attendant while clumsily separating the lids preparatory to the cleansing.

2. *Curative.*—During the first stage of the inflammation, when the conjunctiva is rapidly increasing in vascularity, and the discharge is thin and watery and contains flakes of mucous and epithelium, application of cauterants is to be avoided, and the treatment directed to the checking of the advancing inflammation by ice compresses and the free use of antiseptics and germicides. The ice compresses are to be constantly applied. Small pads of gauze, half an inch thick, cold from contact with ice, are successfully laid on the lids, and the moment their temperature is raised by absorption of heat from the skin they are replaced by fresh ones. The nurses of the Philadelphia Hospital have become expert in this manipulation. I take this opportunity of warmly commending their skill and faithfulness. Antiseptically clad, they devote themselves for many successive hours to their monotonous and arduous, but necessary labor, and have saved many eyes that would otherwise have gone on to destruction. Equally well do they execute the other essential part of the treatment of the first stage, namely, the cleansing of the conjunctival sac. After comparative trial of many of the antiseptics in common use, I have concluded that the best results are secured from the permanganate of potassium. It is used in strengths varying from 1 : 2000 to 1 : 600. I have reported (*Trans. Sec. Opth., Coll. Phys.*, Phila., 1901) four cases of ophthalmia neonatorum in which the effect of the permanganate solution was astonishingly prompt and gratifying. After several douchings of 1 : 1500 solution, the little patients rapidly recovered without corneal involvement. The disease was of average severity in all, and in the secretion of the conjunctival sac the gonorrhœal bacillus was abundantly represented. The single disadvantage of the permanganate solution is the staining of the surface of the lids and conjunctiva a dark purple hue—

a disadvantage offset, however, by its exposure of commencing ulceration of the cornea. Permanganate possesses the quality of staining corneal ulcers, outlining and covering them with a distinctness equal to that obtained from fluorescin.

In the second stage, when the inflammation is at its height, the discharge is yellow and purulent, and involvement of the cornea common, no remedy is as effective as nitrate of silver, ten grains to the ounce. Argyrol and protargol, offered as substitutes, have proven useful, and if one could accept the favorable reports published of their efficacy, quite as useful as the nitrate solution. Unless, however, they can be shown to be superior, I see no reason for deserting the remedy, that has proven to be of the greatest value, for others whose merits are not proven.

A CASE OF GANGRENE OF THE LUNG CURED BY OPERATION.

By DAVID RIESMAN, M.D., A. C. WOOD, M.D.,
AND
G. E. PFAHLER, M.D.

The history of the case is as follows:

I. P., a negro, aged sixty-three years, born in Delaware, was admitted to my wards (R.) in the Philadelphia Hospital on July 22, 1902. His mental condition was such that a complete history was not obtainable; but it was ascertained that his family history was good, and that his own health had always been satisfactory.

The illness on account of which he entered the hospital began four days prior to admission, with chill, pain in the right side, and cough. Examination showed the patient to be an emaciated man, with excessively tortuous temporal arteries, and hard, beaded radials; with arcus senilis and contracted pupils. His skin was hot and dry, the tongue coated, the breath offensive, the temperature 103.6°, the pulse 110, hard and incompressible, and the respirations 36. The chest showed diminished expansion on the right side. The percussion resonance was impaired over the middle of the right lung posteriorly and in the middle of the right axillary region. Over this dull area fremitus was increased. Auscultation did not afford any satisfactory revelations. The heart sounds were feeble, except for the second aortic, which was accentuated.

The temperature remained elevated—between 102° and 103.4°—for two days. During the night of July 24th it fell rapidly from 103° to 98°, five degrees, without any sweat. The respiration dropped to 28; the pulse to 88. Within a few hours, however, by 11 A.M., the temperature had again risen to 101.6°, declining by the following morning at 8 to 97.8°.

Examination at this time revealed very little of moment in the chest. No distinct area of consolidation could be discovered, and judged by the physical signs, the man's condition had greatly improved. The symptoms, however, did not show a corresponding amelioration. Despite the fall in temperature, pulse, and respiration rate, the patient's state appeared grave. He was delirious and struggled to leave the bed, had insomnia, coughed a great deal, and passed urine and fæces involuntarily. During the time in which the quantity of urine was measurable, it was from twenty-two to thirty ounces; it contained albumin and an abundance of hyaline and granular casts, was acid in reaction, and had a specific gravity of 1020. The bowels were loose, greenish in color, and very offensive. There was no enlargement of the liver or spleen.

The temperature remained decidedly subnormal for four days, between 96° and 98°, the highest point in the day being reached at either 8 P.M. or 2 A.M. Only once during the four days did it ascend above 99°. The breath was offensive,

especially during coughing, and on July 28th the resident physician, Dr. Dalby, noticed that the sputum had a gangrenous odor. In a specimen examined at this time I was not able to find elastic tissue or fatty acid crystals. Tubercle bacilli were also absent from the sputum.

An examination of the chest made on July 28th showed the following:

Slight impairment of resonance on the left side on the first and second interspaces in front; a normal note elsewhere over the left chest. On auscultation, the breath sounds were found to be feeble over the entire left side, including the area of slight dulness. Here, however, the expiration was a trifle prolonged, but neither harsh nor bronchial. On the right side nothing was found in front; posteriorly, however, important physical signs were discovered. There was a dull patch to the inner side of the lower half of the vertebral border of the scapula, in the scapulovertebral space, the dulness extending downward and forward to just behind the postaxillary line. Over the dull area, especially in the spinal gutter, both tactile and vocal fremitus were increased. On auscultation, bronchial breathing could be heard over a small area inside of the lower half of the scapula, close to the spine, at the fifth rib. Below the angle of the scapula the breath sounds were normal, but became faint in the axillary region. The pulse was feeble and almost thready, the breath gangrenous. The patient was delirious and had to be restrained. Dr. Wood saw him with me at this time, and agreed as to the advisability of operation. The man was transferred to the surgical wards, and, at my request, Dr. Pfahler made an X-ray examination. Inspection with the fluoroscope showed impaired movement of the right half of the diaphragm, with a distinct shadow at the right apex. The lower portion of this shadow was on a level with the base of the spine of the scapula. A negative was made, which was developed while the patient was being etherized and prepared for operation. This exhibited more distinctly the area involved, and showed a dense wall surrounding a lighter area in the upper part of the right lung. The lower portion of this wall was on a level with the base of the spine of the scapula, and apparently included all the lung space above this point. The patient moved during the exposure, and thus spoiled what would otherwise have been a good negative. Physical examination just prior to the administration of the anæsthetic showed, as before, a small patch of dulness and bronchial breathing in the area between the spine and the right scapula.

Guided by the physical signs and by the information furnished by the radioscopic examination, we were able to determine in advance just where to make the incision and which rib to excise, the fifth being the one chosen.

The following are Dr. Wood's notes of the case:

"I saw the patient referred to in the foregoing report, through the courtesy of Dr. Riesman, on the 28th of July last. The most striking feature was the very offensive odor that was observed upon entering the room, which became more pronounced as the patient was approached. The odor that came from the man's breath was typical of gangrene. The other features that impressed me were the subnormal temperature and the confused mental condition of the patient. The result of the physical examination is fully detailed above. I had no hesitation in coinciding with Dr. Riesman in the diagnosis of gangrene of the lung, and in the opinion that an early operation should be performed. The only point that seemed

in doubt was whether the patient would survive the operation; but, as we felt that recovery was impossible by any other means, we considered it proper to give him this chance.

"The gangrenous area of the lung had been very carefully outlined by Dr. Riesman and Dr. Pfahler; and the latter had taken an excellent skiagraph which fully confirmed the location of the lesion as previously determined by physical examination.

"The operation was performed July 29th. Just before the anæsthetic was administered, Dr. Riesman again, at my request, outlined the area of dulness, which was on the level of the fifth rib, on the right side posteriorly, between the inner border of the scapula and the spine.

"Chloroform was administered until anæsthesia was complete, when ether was substituted. The skin was then cleaned in the usual manner, and the fifth rib exposed by a slightly curved incision, an inch and a half or two inches of it being resected by the subperiosteal method. The visceral and parietal layers of the pleura were found to be adherent and dark and lustreless in appearance. The gangrenous portion of the lung was found to be just beneath the pleura, so that no delay or difficulty was experienced in reaching it. Some fragments of necrotic lung tissue were removed. Digital examination showed the cavity to be as large as a man's clenched fist. It was not thought proper to irrigate the cavity, but the walls were gently cleansed with the aid of gauze sponges, which brought away all loose dead matter. The cavity was then packed with sterile gauze, and the usual dressings were applied.

"The patient bore the operation very well, and did not seem any worse in consequence. The wound was redressed daily, the packing and any sloughs that appeared being removed, after which fresh packing was introduced. The discharge was very profuse at first. The breath rapidly lost its foul odor; and, although the wound remain very offensive for some time, it gradually lost this character. The delirium persisted for some days, but was less and less pronounced, and finally disappeared entirely.

"Instead of a subnormal temperature, which was present before operation, the temperature rose immediately, reaching 101°. The pulse was 112 on the evening of the operation. On the fourth day, the temperature reached 102.6°; pulse, 104. From this time both temperature and pulse gradually declined. Cough continued to be a prominent symptom, but became less troublesome. The general condition of the patient changed for the better in a very satisfactory manner.

"While dressing the wound it was noted that air was expelled violently when the patient coughed, showing that a rather large bronchus opened into the cavity. The volume of air thus escaping from the wound diminished continuously as the cavity healed."

August 25th a note says: "Patient still has cough, but otherwise is in a fair condition. Air still comes through the wound, but the probe shows that the cavity is decreasing in size. The patient feels well and wants to sit up."

From this time on the convalescence was rapid and satisfactory.

Some of the points of interest brought up by this case are: First, the use of the X-ray as an aid in locating the pulmonary lesion. While the procedure is no longer new, another instance of its value is worth

recording. Although the skiagraph confirmed the physical signs accurately, and in no way modified the treatment, it was very satisfactory to have this confirmatory evidence. It must be admitted that physical signs are sometimes misleading, and it is in these cases that the X-ray gives invaluable service. Tuffier (*Revue de Chirurgie*, August, 1901) advises that when skiagraphic findings do not coincide with the results of auscultation and percussion, the latter be ignored and the operation be done according to the evidence furnished by the former. He cites a case in which the skiagraph showed a lesion toward the base of the lung, while the stethoscope indicated a higher seat. The incision was made at the point indicated by the stethscope, with negative results. The next rib below was then resected, and the focus was discovered in the place shown by the X-ray. The same author reports eight cases in which he has applied radiography to pulmonary affections. The results were extremely satisfactory in five instances, and negative in three. In some pathological conditions the X-ray fails entirely, but gangrene of the lung has been found to cast distinct shadows. In accepting the X-ray evidence it is necessary to be assured of the fact that the plate is satisfactorily clear. If doubt exists, it is better to discard it altogether. In order to be as certain as may be, it is desirable always to have two skiagraphs taken, and to note whether they conform with each other.

The importance of definitely locating a lesion of the lung before undertaking any operative procedure will appeal to everyone. On this point Tuffier states that operations on the lungs when the lesion was accurately diagnosed beforehand were successful in 71 per cent. of the cases—that is, that the mortality was 29 per cent.; while the mortality was 60 per cent. in the cases in which there was a mistake in the diagnosis. Of 300 cases collected by this author the lesion was incorrectly diagnosed in 48.

The resection of a rib may be done under local anæsthesia if the condition of the patient will not warrant the use of a general anæsthetic; but in our case the delirious condition of the man made him an unsuitable subject for the former. When a general anæsthetic is necessary, chloroform is usually to be preferred.

The resection of ribs and the pleural opening should be ample in cases of pulmonary gangrene. This is more essential in these cases than in abscess of the lung, as the healing process is slower, and it is

necessary to keep the external opening patulous until the cavity is entirely closed. With a liberal opening, also, the wound may be drained with gauze, which I believe to be more efficient and suitable in these cases than tubes.

When the parietal and visceral layers of the pleura are adherent the lesion may be opened immediately; but all operators agree concerning the desirability of securing this condition before attacking the lesion. When adhesions do not exist, therefore, the two layers should be stitched together around the proposed opening into the lung, which may be made at the end of forty-eight hours, or sooner, if the patient's condition demands earlier relief.

Some operators advise against irrigating the cavity in these cases, and with these I am in entire accord; others are equally convinced of the value of this procedure. Those who hold the latter view should follow the method advised by Cotterill: "The fluid should be a mild antiseptic (not carbolic lotion); it should be warmed to about blood heat; it should be injected slowly and without force, great care being taken that it runs out as fast as it runs in. The patient should be rolled over into such a position that it is impossible for a large volume of the lotion to collect in the chest at one time, and, by its weight, embarrass the heart. . . . The fluids I prefer to wash out with are: boric lotion, a solution of creolin, or a solution of izal, the latter of which is a particularly useful deodorizer."

"Herezel states that about one-fourth of all the recorded operations upon the lung have been for gangrene. Of 91 cases of pneumotomy for gangrene, 60 to 61 per cent. were successful. Garré reports 122 cases operated upon for pulmonary gangrene, of which 66 per cent. were cured. He states that but 20 or 25 per cent. recover without operation."

The patient's present condition is most satisfactory. He feels strong, his mind is clear, and he has become a useful helper about the ward. Physical examination shows a slight depression below the right clavicle. The percussion note is the same on the two sides in front, but is slightly impaired in the right axilla, and below the operative wound for a distance of one and one-half inches. Auscultation shows slightly harsher breathing, and some increase in vocal fremitus in the neighborhood of the wound—but not extending more than an inch above this. The urine has a specific gravity of 1018, and contains a faint trace of albumin, but no casts. The man has gained between thirty-five and forty pounds in weight. There is a slight discharge from the sinus, but the indications are that it will soon be closed.

While the chief interest in this case centres in the happy results of the operation performed by Dr. Wood, there are several points of importance to which brief allusion will not be out of place. The first of these is the cause of the gangrene. The history of an acute onset during fair health, with chill, cough, and pain in the side, suggests that the primary condition was lobar pneumonia. Why this so rapidly —apparently in less than ten days from the time of onset—passed into gangrene, cannot be answered, except in a speculative way. It must be remembered that the patient had pronounced arterio-sclerosis and albuminuria, conditions which, it is admitted by all, favor the development of gangrenous processes in the lung.

Lobar pneumonia is an accepted precursor of gangrene; but Aufrecht,[1] among 1501 cases, did not find it in a single instance—a most remarkable circumstance. He also cites Grisolle,[2] who, among 305 cases of pneumonia, found none followed by gangrene, and who, in seventy cases of pulmonary gangrene, was able to find only five in which the condition had supervened upon pneumonia. Osler,[3] however, observed it three times in 100 cases of pneumonia (3 per cent.), and according to Norris,[4] there were three instances of gangrene in 500 cases of pneumonia at the Pennsylvania Hospital—a proportion of 0.6 per cent. Tuffier,[5] in his collection of seventy-four cases, found pneumonia to be the etiological factor in more than thirty; and Pomeranzew[6] found five cases of gangrene among 727 of pneumonia.

In the only other case of gangrene that has come under my own observation—one that I did not see during life, but upon which I made the autopsy—the condition also followed lobar pneumonia. Other causes of gangrene that need merely be mentioned are embolism, the aspiration of foreign bodies and of putrid material from the upper air-passages, wounds of the lung, tuberculosis, bronchiectasis, and the rupture of an empyema into the lung. Embolic gangrene has been observed after abdominal operations, in puerperal sepsis, as a sequel to middle ear disease, and secondarily to other abscesses and pyæmic foci. The fetid material that is the cause of aspiration gangrene may come from the pharynx; from the œsophagus, in cases of obstruction or rupture; or, as in a case reported by Packard and Le Conte,[7] from a bronchiectatic cavity. Gangrene also occurs as a rare effect of compression by aneurisms or tumors. Diabetes, typhoid fever, alcoholism, and—according to Gee and Herringham[8]—scurvy are predispos-

ing causes of gangrene. It is probable that in the majority of cases of gangrene the system at large or the lung itself was previously in a state of lowered vitality.

The second point that I desire to emphasize is the absence of fever in our case. Fever, according to the majority of writers, is an important symptom of gangrene. Aufrecht[9] states that he has never seen it absent. Usually it is high—40° C. (104° F.) and over. At times it is remittent. As the temperature chart shows, the gangrene in our case pursued its course, not only without fever, but with a subnormal—almost a collapse—temperature. Pulse and respiration were equally unsuggestive of serious trouble, and it was only the man's general condition, his delirium and prostration, that made us feel that he was in grave danger. At the Philadelphia Hospital the opportunities for observing anomalous types of acute pulmonary disease are abundant. Only recently I saw a case of complete pneumonic consolidation of the left lung, typical at autopsy, and complicated with purulent pericarditis, in which there had been no fever.

The third point worthy of emphasis is the character of the sputum. As stated in the notes of the case, there was no elastic tissue, and fatty acid crystals were likewise absent. The odor, however, was characteristic of gangrene. In the diagnosis of pulmonary gangrene the sputum deserves careful attention. In typical cases it has a tendency to separate into three layers, the lowest consisting of globular masses with a tendency to confluence; the highest being frothy, and having masses of dirty sputum depending from its surface. The intermediate stratum is composed of a greater or lesser quantity of turbid, opaque fluid. When a part of the bottom layer is boiled with a 2 per cent. solution of potassium hydrate and examined microscopically on a slide, wavy bundles of elastic tissue are commonly found; but elastic tissue may be entirely absent. This fact is explicable on two grounds: First, the gangrenous process may have been arrested, the cavity no longer spreading; and second, the elastic tissue may have been dissolved by the fermentative action of the fluid—an action that is probably due to bacterial activity. It has been shown by Müller and by Simon, to quote an analogous instance, that the pneumonic exudate is dissolved by a proteolytic ferment. The fatty acid crystals are frequently absent. They are usually found in old cases of gangrene; ours was, of course, acute. In some cases of gangrene the sputum

presents nothing peculiar, and the destructive process in the lung is discovered only at autopsy.

An attempt has been made to differentiate the odor of fetid bronchitis from that of gangrene. Aufrecht compares the former to the smell of stale cheese, and the latter to that of the fluid of manure.

It is scarcely necessary to discuss the other diagnostic features of gangrene. The sputum and the fetor of the breath are, as Osler[10] says, the distinctive features. The important point is accurately to locate the involved area. When the gangrenous process is circumscribed, physical examination usually renders this possible; at least, it serves to focus attention upon a particular area in the lung. When, however, the process is more or less diffuse or the foci are multiple, accurate delimitation is difficult. In the circumscribed form, there may be signs of a cavity or of consolidation. When the symptoms are clear and physical examination gives but dubious results, a careful search should be made for the presence of râles, as their detection may be the only clew to the situation of the trouble. Errors are, however, frequent. Strange to say, the physical signs are likely to locate the focus either too high or too low; the latter is more often the case. Furthermore, they do not give an exact idea as to the depth of the gangrenous area beneath the surface of the lung; and they often fail to map out its actual size. Usually the gangrenous cavity is found to be much larger than was suspected. Gangrene has occasionally been mistaken for empyema, as in a case reported by Körte.[11]

Exploratory puncture naturally suggests itself as an aid in diagnosis and localization; but, on account of the danger of causing infection of the pleura, with consequent empyema or, by carrying the needle that has been infected by its penetration of the cavity through a healthy part of the lung, of producing a spread of the infection throughout the lung, exploratory puncture is better avoided. It is condemned by Lenhartz,[12] by Tuffier,[13] and also by Terrier and Reymond;[14] the last-named authors think it permissible only as a prelude to operation.

As has been pointed out by Dr. Wood, the X-ray is of great value in the diagnosis, as it usually indicates, with fair precision, the size and position of the gangrenous focus. Depending upon whether the cavity is full or empty, there will be either a dark shadow or a light area in the affected region of the lung. In addition, the X-ray will show a

lessened mobility of the diaphragm on the diseased side. I would suggest that in hospitals in which radiographic examinations can be made with facility, every case of acute lung trouble in which abscess or gangrene is a possibility be subjected to such an examination.

Prognosis.—In acute forms of gangrene the prognosis is better than in the chronic. Metapneumonic gangrene has a decidedly larger percentage of recoveries than has gangrene due to embolism, aspiration, or bronchiectasis. In Tuffier's table of 71 cases, showing the relation of etiology to results after operation, there are 55 cases of gangrene following inflammatory affections of the lung, with 39 recoveries. There were 4 cases of bronchiectatic gangrene, with 1 recovery; 2 due to foreign bodies, with 1 recovery; 7 of the embolic variety, with 2 recoveries; 1 due to chest wound, with recovery; and 2 following perforation of the œsophagus, both ending fatally.

The mortality from gangrene without operation is necessarily very high—between 70 and 80 per cent. (Verneuill,[15] 80 per cent.). Under operative treatment the death rate falls to 34 per cent., according to Garré and Sultan,[16] who have collected 122 cases, with 80 recoveries and 42 deaths. In Eisendraht's[17] series of 28 cases of pneumotomy for gangrene there were 20 recoveries, 2 ameliorations, 6 deaths. One interesting feature alluded to by several writers is the apparently complete restoration of the diseased lung. In some cases reported by Lenhartz[18]—who has operated upon 25 patients for gangrene, 13 of whom recovered—physical examination and examination with the X-ray showed perfectly normal lung where previously there had been a large and gangrenous cavity. That author gives it as his opinion that in such cases there must have been a re-formation of lung tissue. The second radiograph taken of our case also shows a restored lung. Whether there has been a true hyperplasia of air vesicles or merely an enlargement of the existing ones, it is not possible to say without experimental data.

All writers upon the operative treatment of gangrene refer to the importance of adhesions. Their presence greatly enhances the chances of a good result; their absence predisposes to pneumothorax and to the entrance of septic material into the pleura, with resulting empyema. Lenhartz advises that if the condition of the patient does not demand immediate pneumotomy, this operation should be performed in two steps, the lung being fixed to the chest wall at one time, and opened

later. When, however, the signs indicate that procrastination would be dangerous, it is best to open the lung without delay, care being taken to draw the organ into the wound and to stitch it, so as to guard against the occurrence of pneumothorax and empyema.

List of References.

[1] Aufrecht. Nothnagel's Specielle Pathol. u. Therapie, Bd. xiv.; Die Lungenentzündungen, 422.

[2] Grisolle. Quoted by Aufrecht, p 421.

[3] Osler. Practice of Medicine, fourth edition, 130.

[4] Norris. American Journal of the Medical Sciences, June, 1901.

[5] Tuffier. Chirurgie des poumons, 1897.

[6] Pomeranzew. Chirurgie (Russian), v., 301; Centralblatt f. Chirurgie, 1900, 312.

[7] Packard and Le Conte. American Journal of the Medical Sciences, 1902, cxxiii., 375.

[8] Gee and Herringham. Allbutt's System of Medicine, vi., 598.

[9] Aufrecht. Loc. cit.

[10] Osler. Loc. cit.

[11] Körte. Freie Vereingung d. Chirurgen, Berlin, February 11, 1901; Centralblatt f. Chirurgie, 1901, 380.

[12] Lenhartz. Mittheil. aus den Grenzgebieten d. Medicin u. Chirurgie, 1902, ix., 358.

[13] Tuffier. Loc. cit.

[14] Terrier and Reymond. Chirurgie de la plèvre et des poumons, 1899; Centralblatt f. Chirurgie, 1900, 979.

[15] Verneuill. Ann. de la Soc. Belge de Chirurgie, 1900, viii., 148.

[16] Garré and Sultan. Beit. z. klin. Chirurgie, 1902, xxxii., 492.

[17] Eisendraht. Philadelphia Medical Journal, 1901, viii, 786.

[18] Lenhartz. Loc. cit.

COAL-TAR AND ITS DERIVATIVES IN THE TREATMENT OF DISEASES OF THE SKIN.

By M. B. HARTZELL, M.D.

Although a large number of substances derived from coal-tar have at various times been recommended for the treatment of diseases of the skin, the number actually in use at the present time is a relatively small one, since the great majority of them, when brought to the test of extended use, have failed to justify the more or less extravagant claims of enterprising manufacturers or enthusiastic, and oftentimes too credulous, investigators. Of those in present use only a small number can be said to be remedies of choice, the remainder being employed as a sort of last resort when the older remedies fail.

The coal-tar products possess, among other properties, two which suggest their use as local applications in many diseases of the skin: they are nearly all more or less inimical to the lower forms of life which play a prominent rôle in a number of cutaneous diseases, and they are analgesic to a greater or less degree when applied locally or taken internally; and these two properties represent their chief claims to employment in dermatology.

I shall not attempt in this short paper to enumerate all the derivatives of coal-tar which have been employed in the treatment of diseases of the skin, but shall very briefly refer only to those which may be said to have established a reasonable claim to consideration by relieving more or less effectually some of the troublesome symptoms of various cutaneous maladies.

As *liquor carbonis detergens*, *coal-tar saponine*, *coal-tar Le Bœuf*, coal-tar itself has been used for some time in the treatment of diseases of the skin with success, especially in those cases in which the ordinary wood-tar has been found useful, such as chronic eczema with

r. In this disease it diminishes hyperæmia
the itching, which is so frequent and
ed, in the opinion of no less an au-
son, it is the most generally useful

of all the remedies employed in this affection; and Leistikow declares that it relieves itching as no other remedy does. The latter finds the simple alcoholic solutions more effective than the combination with tincture of quillaia, which is used in the preparation of the liquor carbonis detergens, and employs the following formula:

℞.—Olei lithanthracis, 30.0 . . . . . f℥j.
Alcohol (95 per cent.), 20.0 . . . . . f℥vij.
Ether, 10.0 . . . . . f℥iiss.—M.

My own experience with this remedy leads me to value it highly in the treatment of chronic eczema, and to a less degree in psoriasis. It may also be used as an ointment, the best vehicle for this purpose being, according to Leistikow, the *unguentum caseini* of Beiersdorf.

Carbolic acid, one of the earliest employed of the coal-tar products, is one of the most generally useful of the entire series on account of its anæsthetic effect when applied to the skin. Although an active parasiticide, it is not much employed in the treatment of parasitic affections because of the introduction of more active and more agreeable remedies of this class. Because of its benumbing effect when applied to the unbroken skin it finds daily use in the treatment of that very large class of skin affections in which itching is a more or less prominent symptom. As an antipruritic it probably has no equal in the whole Pharmacopœia, and on this account is well-nigh indispensable to the dermatologist. It may be used either as a lotion or ointment in every disease in which it is desired to relieve itching, alone or combined with other remedies. The following lotion is an admirable one for the relief of itching, whatever its origin:

℞.—Acid. carbolic. . . . . f℥ss.
Glycerini . . . . . f℥j.
Aq. camphoræ . . . . . q. s. ad f℥iv.—M.

The following ointment, in which the carbolic acid is combined with an equal quantity of camphor, will be found an excellent application in many itching diseases:

℞.—Acid. carbolic., } . . . . . āā gr. xv.
Camphoræ, }
M. et adde
Lanolin., } . . . . . āā ℥ss.—M.
Petrolat., }

As an antipruritic there are few or no contraindications to the use of carbolic acid.

Savill has reported a case of leucoderma, occurring in a girl sixteen years old, in which pure phenol painted upon the patches caused the skin to resume its normal pink color in three weeks.

The *cresols*, which are closely akin to carbolic acid in their chemical composition, have been used to a limited extent in cutaneous diseases as *creolin* and *trikresol*, the former probably being cresol emulsified with resin soap. Creolin may be used in the same class of diseases in which carbolic acid is useful, but according to my own experience it not infrequently proves irritating, especially when used as an ointment.

Trikresol has been highly recommended by McGowan in the treatment of alopecia areata; and more recently Heidingsfeld, who has used a 50-per-cent. solution painted over the bald areas, has reported excellent results. The latter believes it the best of all applications in the treatment of this frequently obstinate disease. In several cases comparative experiments were made with this remedy and some of the older ones, and it was found that the growth of hair was much more rapid in the patches treated with trikresol.

Although first obtained from galbanum by Hlasiwetz and Barth, *resorcin* properly belongs among the substances derived from coal-tar, being a diatomic phenol possessing physiological properties resembling those of carbolic acid. Some years ago I called attention to the many valuable properties possessed by this drug in the treatment of diseases of the skin, and a longer and more extended experience in its use has only confirmed the opinions then expressed. Used as an aqueous lotion, in the strength of 8 to 10 grains to the ounce, it is an extremely valuable application in many of the forms of eczema, especially in that variety which has been recently designated seborrhœic eczema, allaying itching, diminishing hyperæmia and discharge. For some reason, about which I am not very clear, it does not seem to answer nearly so well in this affection when employed as an ointment. In the treatment of acne it has no superior, used as an ointment in the strength of 40 to 60 grains to the ounce, or even stronger. Although formerly inclined to regard it as inferior to sulphur in the treatment of this disease, a more extended experience leads me to give it first place. In superficial epithelioma, such as is seen so frequently upon the face, resorcin sometimes acts most happily in promoting cicatrization, used as a 40-

to 50-per-cent. plaster. A weaker plaster—20- to 25-per-cent.—may be used to promote healing after the destruction of such growths by stronger caustics, such as caustic potash or pyrogallol. Although a fairly active parasiticide, it is rarely used for this purpose at present. Resorcin possesses decided sedative properties, and it may be used with advantage in the treatment of painful chronic leg ulcer; I know of no remedy which affords the same relief in this notoriously intractable disease.

Naphtol, also known as *beta-naphtol*, another phenol derived from coal-tar, possesses parasiticide properties much more active than the phenols already considered. First proposed by Kaposi as a remedy for scabies, it has proven itself a very satisfactory substitute for the drugs usually employed in the treatment of this disease. It is practically without odor, and does not stain, and although it sometimes causes sharp burning when first applied, it does not produce a dermatitis such as often follows the use of sulphur. Within the past two years I have used it quite extensively in that very obstinate parasitic affection, ringworm of the scalp, and have found it more efficacious than the parasiticides more commonly used. It rarely causes any irritation of the scalp, even when used in ointments containing from a drachm to a drachm and a half to the ounce. Apart from its employment as a parasiticide, this drug is but little used in the treatment of diseases of the skin. It has been recommended as a local application for psoriasis, but it is much less useful than a number of other substances, and is not likely to find extensive employment in this affection.

Orthoform, a recently introduced coal-tar derivative, at first promised to be a very useful remedy in the treatment of painful leg ulcers and other painful affections of the skin in which the terminal nerve-endings are exposed, since its application to the broken skin is followed by marked anæsthesia lasting several hours. This promise of usefulness, however, was not fulfilled, since it was soon found that, while it produced a decided and lasting local anæsthesia, its local use was followed in a considerable number of instances by a painful dermatitis, and even in a few cases by gangrene of the skin. My own experience with it was decidedly unfavorable.

Acetanilid, pure or diluted with some inert powder, such as oxide of zinc, talc, or starch, has been found useful as a dressing in ulcers and burns, relieving pain, preventing suppuration, and promoting cicatri-

zation. While possessing undoubted antiseptic and sedative properties, it has the serious disadvantage that it sometimes produces decided toxic effects, as shown by cyanosis and symptoms of collapse, through absorption, even when applied to a comparatively limited area. While no doubt useful, it has not come into general employment.

Epicarin is a recent addition to the list of parasiticides derived from coal-tar, which is said to be non-toxic and unirritating. It has received the indorsement of Kaposi as an efficient remedy for scabies. Further experience is needed to show whether this remedy deserves a permanent place in the armamentarium of the dermatologist.

Some of the aniline dyes, such as *pyoktanin* or *methyl blue*, *methylene blue*, and *fuchsin*, have been used with asserted good effect in simple and malignant ulcerative diseases of the skin. Elliot has employed an ointment containing from one to five grains of fuchsin to the ounce in Paget's disease, and found that it not only relieved pain, but promoted cicatrization of the ulcerated area. I am not aware, however, that other observers have obtained such favorable results.

A small number of drugs of coal-tar origin belonging to the group of analgesics have been used internally with a considerable degree of success in the treatment of diseases of the skin, chiefly those in which itching or pain is a prominent symptom. Arnstein and Antoniak have reported two cases of chronic pruritus in which the internal administration of *antipyrin* acted most favorably. In the first case the disease had lasted four months, but disappeared in ten days when antipyrin was given twice a day. The second one was a case of senile pruritus, often an obstinate and distressing affection; in this case moderate doses of antipyrin given four times a day caused the disease to disappear in two weeks.

Phenacetine and *acetanilid* have given me excellent results in chronic urticaria, seven or eight grains of the former, or four or five grains of the latter with an equal quantity of bicarbonate of soda, being given four times a day.

From the foregoing it is evident that the coal-tar drugs form a small but important addition to the therapeutics of diseases of the skin. While a large proportion of those presented for trial, when weighed in the balance of experience, have been found wanting, a respectable minority have found a permanent place in dermatotherapy, and are in almost daily use.

EPIPHENOMENA OF CEREBRAL HEMORRHAGE.[1]

By F. SAVARY PEARCE, M.D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASES, MEDICO-CHIRURGICAL COLLEGE OF PHILADELPHIA; NEUROLOGIST TO THE PHILADELPHIA AND TO THE HOWARD HOSPITALS.

By the prefix "epi" we refer broadly to additional or secondary phenomena in the course of disease, and in particular to disease as indicated by the title—Cerebral Hemorrhage—around which there hovers a degree of inaccurate tracing of symptomatology from the earliest pathogenesis to the apopletic attack itself; and, indeed, in the remote symptoms following paralysis. It is to emphasize the due recognition of the apoplectic state by the physician also the insistence upon prophylaxis by him, and by the patient, so that this serious disaster to the central nervous system may be the more frequently prevented, that the subject is brought before this society.

If this lesion, which is constantly growing among active Americans, is to be at all lessened, it will be necessary to enforce essentials for your consideration, and this will entail the rehearsing of a considerable number of facts, well known, but frequently unheeded.

Heredity.—There can be no doubt that hemorrhage into the brain has its hereditary side, and this should be studied closely in families in which such disease has been found, that we may be able to enjoin caution upon the succeeding generations so predisposed—if we may use so strong a term. Therefore, in persons after forty who give a family history of apoplexy, it is important to guard the arterial system in every way. The vulnerability of the vascular system in these persons is too patent to need defense; we simply wish to urge the necessity for recognizing the facts, since treatment *now* is of vital importance. The anatomic side to apoplexy needs careful study in regard to stature, for undoubtedly a person with a short neck and sanguineous temperament is more liable to rupture of the lenticulostriate artery, for example, than one who has small bloodvessels, a long neck, and is of less plethoric diathesis.

[1] Read by invitation before the Lebanon County (Pa.) Medical Society, May 13, 1902.

Prodromes.—In a man of forty, who has repeated attacks of dull headache (" throbbing " in nature) associated with a florid type of constitution, no matter whether pulse tension is high or not, we have reason to believe that the vasomotor system in the brain is at fault; paretic dilation of the bloodvessels is occurring and the " breaking strain " is nearly reached on many occasions, particularly after excesses in eating or drinking. We have all frequently seen cases of the following type:—A man of good physique overindulges in food or alcohol and then complains of headache and sick stomach next day, both of which are indices to the scientific medical man for a crisis that will come in due time with these oft-repeated congestions.

It was Dr. Charles E. Dana who made the statement last year that the average limit for any human body is 3,000 alcoholic intoxications. I am certainly convinced that *one* or a *hundred* intoxications are too much for the normal function of the brain and its circulation, and that each time the brain becomes so engorged we have tendency toward cerebral hemorrhage. No physician should doubt this who reads upon the face of a debauchee that grosser sign of a congested facial dermis, the aftermath of a " night out." It is a sad picture to me personally, for I seem to see the microscopic pathology going on in the highest structure of the body—the nerve cell—and the resultant malnutrition of the vasomotor system, which in turn permits arteriosclerosis and fatty change in the vessel walls, tending toward the sure approach of apoplexy. I do not wish to moralize or preach a sermon, but simply to give the scientific facts of the case of the surrounding (" epiphenomena ") signs of apoplexy. That one person undoubtedly resists the physical and mental effects of acute alcoholism more than another is true; but I can find no statistics to oppose the fact that these same individuals prone to excesses are more invulnerable to arteriosclerosis, inflammation of serous membranes, and liability to autointoxication from disturbed metabolism—in fine, an increasing vicious pathologic circle with premature senility or early death from apoplexy. How many times have you seen the " healthy " man, described by the laity and quietly presumed to be so by the physician, carried off suddenly by heart failure, so-called, cerebral congestion, thrombosis, embolism, or apoplexy? *No such man was well, in scientific terms, for months or years preceding his demise!*

We wish to emphasize the points rehearsed above, for it is here alone

that degeneration of the nervovascular system may be prevented; after the typical prodromes present, which we all know so well, the danger line has been surely passed. When vertigo, a high blood-count of the red cells, catarrhal gastritis, congestive headaches, forgetfulness, and hardened arteries are combined, we may or may not save the patient by conscientious statements of the facts to him, although we are exempted from blame if he does not submit to the treatment we will surely recommend and carry out for the just-mentioned symptom complex. As to treatment here the abandonment of venesection at the present day is to be regretted, for I have seen in my own practice cases in which the patients who were plethoric and presented the signs just rehearsed were greatly relieved upon withdrawing a pint or more of blood, and the apopletic attack was undoubtedly prevented. Of course, all other methods should be enjoined, such as attending to the emunctories, especially the kidneys, and to be on the sharp lookout for insidious nephritis, a malady frequently simulating apoplexy.

Epiphenomena of Attack.—Some other associated symptoms in the attack of apoplexy itself are worth while emphasizing—*e. g.*, the great arhythmia of the heart and disproportionate high temperature on the paralyzed side of the body in cases of hemiplegia following apoplexy; also the continuance of the extreme mental depression or irritability with lachrymosis predominating as important phenomena indicating a greater gravity of the case. In these cases it seems to me that the trio of disproportionate prominent symptoms mentioned only show the profound affection of the nervous system, whether it be due to size or extent of lesion or to the vitality and resisting power of the patient. Another important point in prognosis, even when neither the "attack" or motor palsy of hemiplegic type are prominent, is that if sensory paralysis persists on the palsied side we have a surer indication of greater extent of lesion at the parietal lobe, a greater disorganization of the cerebrum, therefore, and the chances of fatality greatly increased. Another very important outstanding symptom to be looked for both in the attack of apoplexy and following the paralysis is the occurrence of acute or chronic Bright's disease, from the diagnosis of which the disabled cerebrum may be thus the more damaged and death supervene if the kidneys fail, which they are much more liable to do than in Bright's disease alone. I should like to speak also of attacks of pseudoapoplexy due to nephritis alone, of which I have

seen several cases at the Philadelphia Hospital this winter. We found a definite nephritis and a definite hemiplegia with the history of an "attack" so-called. At autopsy there was found absolutely no organic lesion of the brain, although mental symptoms of aphasia and hemiplegia point positively to cerebral intoxication on one side of the brain, due no doubt to combined toxæmia of the cortical neurons and the "wet-brain" or œdema from Bright's disease. Such a condition may occur in other parts of the body. The urine is to be carefully studied, therefore, as an epiphenomenon in apoplexy, since in an irregular attack accompanied by Bright's disease the prognosis of the paralysis will be good in proportion as the nephritis will be amenable to curative treatment.

We have a case under treatment at the present time. The patient is a woman who two years ago had an attack of hemiplegia on the left side, from which she recovered absolutely in a few months. Two weeks ago she developed hemiparesis on the right side of the body accompanied by motor aphasia; the urine was laden with granular casts and albumin. This palsy has also about disappeared, although the progress of interstitial nephritis is well advanced and the pericardium inflamed from the uræmic poison.[1]

The occurrence of hyperpyrexia following in the late clinical course of cerebral hemorrhage is frequently due to disturbance of the heat regulating centres in the corpus striatum when not due to a bronchopneumonia from which so many perish. (See accompanying chart illustrating this point.) In this case it will be seen the temperature mounted to 107° F., and exacerbations to 104° and 103⅘° occurred on two separate occasions.

We would like to mention the possibility of the X-ray being a favorable adjunct toward determination of blood clot within the brain or not as a point in diagnosis between hemorrhage or thrombosis and this confusing class of Bright's palsies. In a case coming to autopsy at the Medico-Chirurgical Hospital ten days ago, Dr. M. K. Kassabian had been fortunate enough to find what he thought was a "shadow" of the thrombotic area in the left lenticulostriate area region, which proved to be so at the postmortem examination. In this case, how-

[1] Since writing this paper the woman died. The pathological conditions described were found, and in addition a small hemorrhage into posterior part of internal capsule and optic thalamus, proving associaton of nephritis and cerebral hemorrhage.

ever, there was no complication of nephritis in making the clinical diagnosis.

Epiphenomena Following Apoplexy.—No matter how apparently well the patient becomes after cerebral hemorrhage, it should be enforced

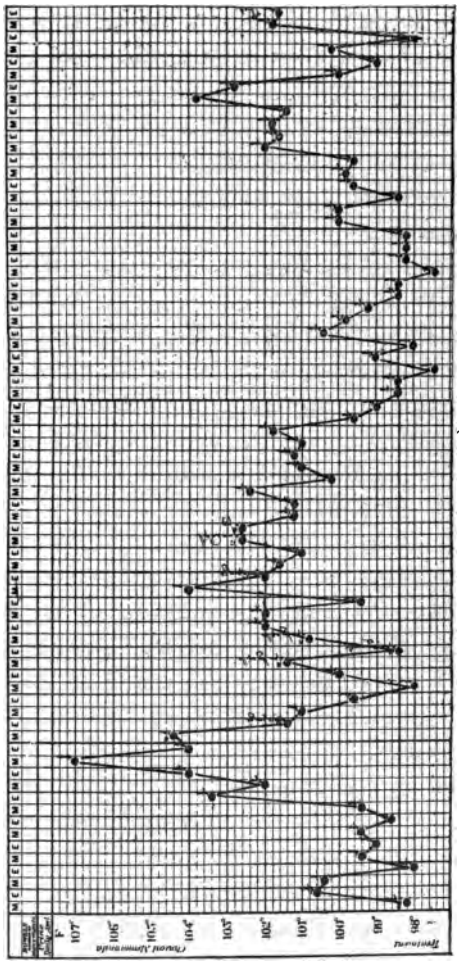
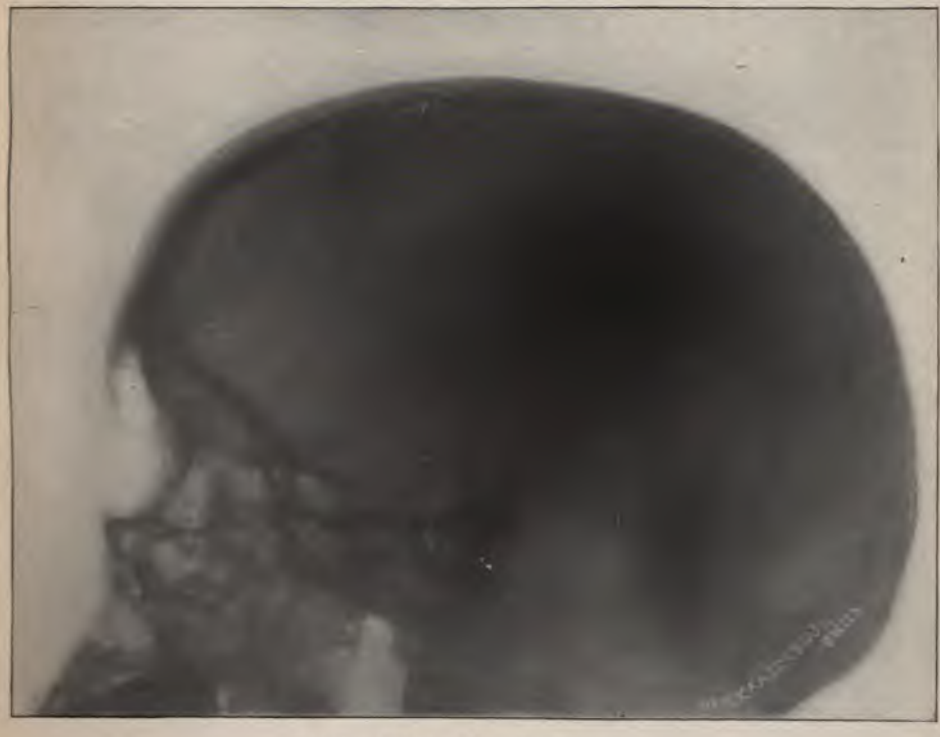


FIG. 1.

that he is a disabled person and should be most cautious in conserving energy, especially brain work. I have seen cases brought to an untimely end by the endeavor to keep at the regular employment of the mind, thus producing acute neurasthenia, symptoms of irritability, and finally early progressive demetia. It is needless to emphasize the

FIG. 2.



Showing shadow beneath motor area.

regime to be carried out in the average postapoplectic state, except to recall the necessity for undue care when the muscular element of the heart sounds is enfeebled, indicating fatty change, or when the endocardium is inflamed; or universal arteriosclerosis exists.

I beg to remind you of the great necessity for interrogation as to specific disease, since these are the most fortunate cases in prognosis through the persistent use of the iodides and mercury bichloride. The sorbefacient effect of the iodides is of much value, even in cases of thrombosis, embolism, or acute hemorrhage, and when the pulse tension remains high with fairly good bodily nutrition. Occasional venesection will be of value, too, in preventing relapse, the possibility of which is never to be overlooked, but attention may be given with decreasing frequency until the fifth year after the "stroke." The patient with fairly good arteries and nutrition passing this time limit will probably survive many years with the hemiplegic disability, providing the greatest care in hygiene is maintained. The frequent use of Kissengen or Carlsbad salts for these persons is of great value through its depleting effect upon the overloaded bloodvessels. The use of nitroglycerin is also of value, as is static electricity through the stimulating effect upon the vasomotor system and the consequent relief of embarrassment to the circulation. The painful condition in the paralyzed parts in postapoplectic patients, which is due to an arthroneuritis, can also be much relieved by massage, hot baths, and the Swedish movements, accompanied by the long spark of the static current.

There are many other points which might be mentioned as epiphenomena of importance in the disease under discussion, but those already discussed seem of the most practical value to bring to your consideration.

If this contribution will have some influence toward encouraging study of detail in individual cases as a means of ameliorating this most unfortunate condition I will be repaid for the endeavor. We firmly believe that only by such study alone will the percentage of cases be reduced and amelioration of the attack be obtained.

PARTIAL PARALYSIS OF ONE UPPER LIMB, RESULTING FROM A VASCULAR LESION OF THE LATERAL COLUMN AND ANTERIOR HORN ON THE CORRESPONDING SIDE OF THE SPINAL CORD.[1]

By WILLIAM G. SPILLER, M.D.,

ASSISTANT CLINICAL PROFESSOR OF NERVOUS DISEASES AND ASSISTANT PROFESSOR OF NEUROPATHOLOGY IN THE UNIVERSITY OF PENNSYLVANIA; NEUROLOGIST TO THE PHILADELPHIA HOSPITAL;

AND

THEODORE H. WEISENBURG, M.D.,

INSTRUCTOR IN NERVOUS DISEASES IN THE UNIVERSITY OF PENNSYLVANIA; ASSISTANT NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

Paralysis of one limb in an adult from a vascular lesion of the spinal cord is of such unusual occurrence that the case we report, in which this condition was present, seems worthy of publication, even though the clinical history leaves much to be desired. Spinal monoplegia, unless resulting from poliomyelitis, seems to be almost unknown. As in our case the lesions were chiefly in one lateral column, and the implication of the anterior horn of the same side seems to have been in large measure secondary to these, it is questionable whether the case could be regarded as one of poliomyelitis.

One other case in literature has a resemblance to ours, and that was a case of congenital spastic rigidity of the limbs, the so-called Little's disease, reported by Dejerine.[2] The lesions of the spinal cord were vascular in origin, and were very similar to those in our case, but were on both sides of the spinal cord, whereas in our case they were unilateral.

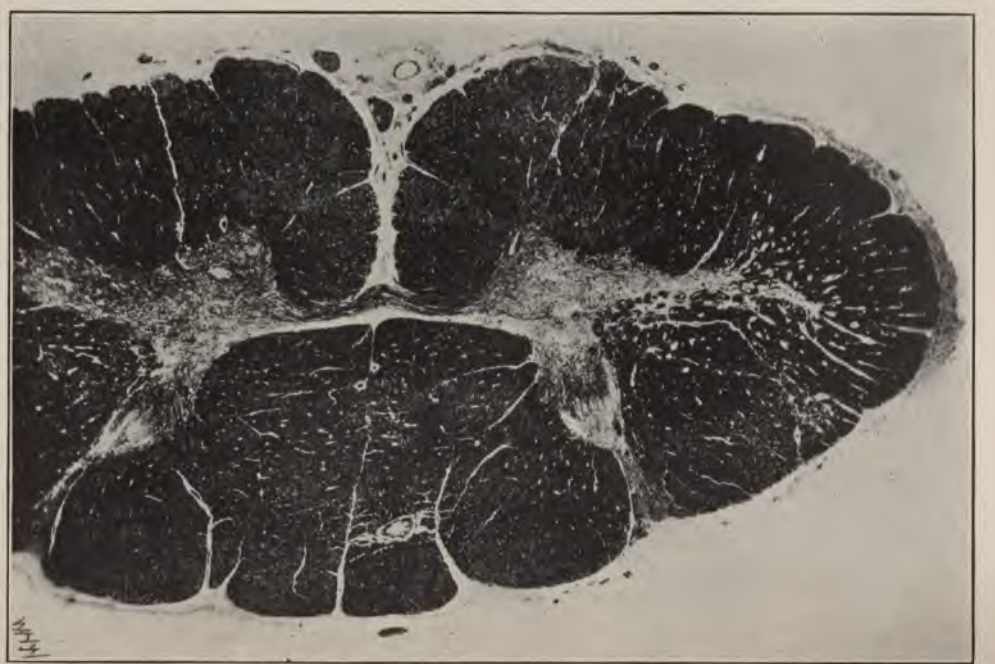
The patient was a man, aged sixty-six years, and was admitted to the Philadelphia Hospital, January 15, 1902. He came later into the service of one of us (Dr. Spiller), but died before a thorough examination of his condition could be made. The ward notes recorded by the historian and resident physician state that he had had typhoid fever, rheumatism, and erysipelas, and had used alcohol freely, but had not had syphilis.

About three years before admission he is said to have had an attack in which

[1] From the Philadelphia Hospital and the William Pepper Laboratory of Clinical Medicine Phœbe A. Hearst Foundation.

[2] Bul. de la Soc. de Biol., 1897, p. 261.

FIG. 1.



Photograph of a section from about the first thoracic segment. The right anterior horn is much smaller and contains fewer nerve fibres than the left. The sclerotic bloodvessels in the right lateral column are shown.

FIG. 2.

Photograph showing the right anterior horn and the right lateral column under higher magnification. The rarefaction of the lateral portion of the anterior horn and the sclerotic bloodvessels in the right lateral column are shown.

speech was lost two weeks. Inasmuch as the loss of speech was temporary, and no signs of a lesion either recent or old were found in the brain, and the man was aged, it is possible that the attack was uræmic in character.

Notes made January 11, 1902, state that during the three days previous the man had been unable to use his right upper limb. He had been employed addressing envelopes. Two weeks before this time he had had a fall, striking his right shoulder, on the ice. Some discoloration followed, but no loss of function.

The tongue on January 11th was protruded straight, and station and gait were good. The knee-jerks were slightly exaggerated. Ankle clonus was not present. The notes also state that the weakness of the right hand was first noticed by the patient one morning on awakening. He was unable to lift anything, and the hand felt cold. Two months after the paralysis of the upper limb occurred some return of power was observed in this limb.

The history unfortunately is very incomplete, and it is uncertain whether the paralysis of the right upper limb developed gradually or not, although it seems to have been of sudden development. The man had had a fall, but it is important that about eleven days are said to have elapsed after the fall before weakness of the upper limb was noticed, and that when the upper limb was paralyzed station and gait were good.

The case while in the hospital was supposed to be one of paralysis of the right upper limb, possibly from a lesion of the brachial plexus. The limb was almost in a natural position at the side of the body; the movement at the shoulder-joint was very limited, that at the elbow-joint was fairly good, and the man was able to place his right forearm over the abdomen and to move the fingers fairly well. The forearm was slightly flexed. He was able to touch his face with his fingers. The condition of the spinal cord would indicate that the paralysis of the upper limb had existed probably more than a few months, as no degeneration by the Marchi method was found, and the degenerative changes in the spinal cord were unquestionably of long duration. The most important statement in this history is that the man had weakness of his right upper limb, and it is uncertain in what way it developed.

The area of primary alteration was in the eighth cervical and first thoracic segments. The blood-vessels were sclerotic within the portion of the right lateral column adjoining the anterior horn and as far as the periphery of the cord. The walls of the vessels here were much thickened, and when stained with acid fuchsin had a glassy appearance (Fig. 1). The neuroglia surrounding many of these vessels near the right anterior horn was much proliferated, and many of the neurogliar fibres were cut longitudinally in transverse sections of the cord. One blood-vessel in the right lateral column had a greatly thickened wall. The lateral and median portions of the right anterior horn were rarefied and stained more faintly than the rest of the horn. The Weigert hæmatoxylin stain showed a great reduction in the number of medullated nerve fibres within these portions of the right anterior horn, and the antero-posterior diameter of this horn was much less than that of the left anterior horn (Fig. 2). The lateral portion of the right horn contained scarcely any medullated nerve fibres, and the right posterior horn also contained fewer fibres than did the left posterior horn. The nerve cells had entirely disappeared in the portion of the right anterior horn where the alteration was most intense—*i. e.*, in the lateral portion, and were very abnormal in appearance where the alteration was less severe.

A small area of slight proliferation of the neuroglia was seen in the columns of Goll along the posterior septum, but did not extend very far upward or downward.

No signs of meningitis were seen, and the blood-vessels of the pia were only moderately thickened.

At a little higher level of the cord, about the seventh cervical segment, the right anterior horn still contained distinctly fewer medullated nerve fibres than did the left anterior horn, and the extreme external portion of the right horn was almost free of nerve fibres, those present having a longitudinal course in transverse sections of the cord. A distinct narrow area of sclerosis was seen extending from the lateral portion of the right horn about half way across the lateral column, and most of the nerve fibres coming from the right lateral column and passing to the right anterior horn through this area had been destroyed. The nerve cells in the lateral portion of the right anterior horn were few in number; some were enlarged and had eccentric nuclei. The right anterior roots obtained in these sections did not appear to be degenerated, but very few roots were obtained.

Sections from the lower cervical region stained by the Marchi method showed no recent degeneration.

Above the lower part of the cervical swelling and below the first and second thoracic segments the sclerotic area became less intense and gradually disappeared.

Sections from the upper part of the cervical region and from the midthoracic region appeared normal. It is, therefore, impossible that this sclerosis could have been caused by a lesion higher than the cervical region.

The destruction of the lateral portion of the right anterior horn is sufficient to explain the brachial palsy, because the cells in this portion of the horn are probably the most concerned in voluntary motion and in the preservation of the muscle substance.

Among recent writers who have mentioned degeneration especially marked in the antero-lateral group of cells of the anterior horns occurring in cases of pronounced muscular atrophy in man, Stanley Barnes,[1] Mott and Tredgold,[2] may be mentioned. The former, in a case resembling one of multiple neuritis with intense atrophy of the hands, found that at the level of the seventh cervical segment the antero-lateral group of cells was the one most markedly affected, only a few dark-staining, shrunken cells remaining; in the other groups of cells little acute change was present, and in the main they were normal.

Mott and Tredgold found a similar condition in two cases of amyotrophic lateral sclerosis. The changes of the anterior horn cells of the cervical region in one case, they say, did not affect all the cell groups; the mesial-anterior group was practically normal, and the change was most marked in the lateral and postero-external groups; within these groups there were scarcely any large ganglion cells having a healthy

[1] Brain: vol. xxv., Winter, 1902, p. 499.
[2] Idem., pp. 405. 411.

appearance. In regard to the other case, they say that the diminution in the number of the anterior horn cells was practically confined to the lateral and postero-external cell groups, the mesial-anterior group showing scarcely any change.

Bikeles and Franks,[1] after cutting certain nerves of the brachial plexus in animals, found by the Marchi method degeneration of nerve fibres within the anterior horn of the cervical region on the operated side. These degenerated nerve fibres could be traced easily toward the lateral group of nerve cells, and none were found passing toward the medial group. They studied also the nerve cells by the Nissl method, in order to determine what groups of cells were degenerated after resection of the peripheral nerves. The most important group of cells in the anterior horn of the cervical region are the ventro-medial, the ventro-lateral, the dorso-lateral, and the central. The names indicate the situations of these groups. In all their experiments on the dog, the ventro-medial, the central, and the ventral part of the lateral group were free from degenerated nerve cells, as shown by the Nissl method. The alteration in all cases was confined to the dorso-lateral group.

These experiments are very valuable, and in connection with the observations on man seem to indicate that the nerve cells situated laterally and dorsally in the anterior horn are the most important in regard to motor function, and it was these cells in our case which were directly implicated in the sclerotic area of the lower portion of the cervical region, so that we have in the microscopic examination of this case reason to believe that the brachial monoplegia was probably of spinal origin.

[1] Deutsche Zeitschrift für Nervenheilkunde, vol. xxiii., Nos. 3 and 4, p. 205.

A CASE OF INTERNAL HEMORRHAGIC PACHYMENINGITIS, IN WHICH THE HEMORRHAGIC CYST WAS OF VERY LARGE SIZE.

By WILLIAM G. SPILLER, M.D.,
NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

It is not my purpose to discuss here the etiology of internal hemorrhagic pachymeningitis, as I[1] have, in association with others, written on both the internal and external forms of chronic inflammation of the dura. The case which I wish to report at this time I have already reported[2] in a condensed form, but without the illustration. The hemorrhagic cyst was the largest I have ever seen within the cerebral dura, and the illustration is desirable in order that an idea of its appearance may be conveyed to the reader. The photograph was made for me by Dr. A. R. Allen. The specimen is deposited in the Museum of the Philadelphia Hospital. The case was one of right-sided hemiplegia.

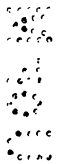
The patient, a white man, aged seventy-three years, was admitted to my service in the Philadelphia Hospital, August 23, 1901. His family history could not be obtained. The man had a slow speech, and difficulty in finding words to express himself; his memory was much affected, and he did not answer questions very intelligently. He remembered that as a boy he had measles, typhoid fever, and scarlet fever. He was unable to say how long he had been sick, or how he had come to the hospital. He made no complaint of pain. He moved his right lower limb with difficulty, and his right upper limb was paralyzed. He had incontinence of urine and fæces. His iritic reflex to light was much impaired. The tongue was protruded with difficulty. The patellar reflex on each side was present, but not exaggerated, and Babinski's reflex was not obtained. The left upper and lower limbs were moved voluntarily and freely while the patient was in bed. The muscles were not atrophied. Passive movement of the right upper limb caused an expression of pain. When the man was made to stand he stood chiefly on the left lower limb, and when made to take one or two steps he dragged the right lower limb. The discomfort expressed by his face, when his right upper and lower limbs were stuck with a pin, showed that the sensation of pain was preserved.

The patient became gradually more stuporous, and granular casts and albumin

[1] Spiller and McCarthy: The Journal of Nervous and Mental Disease, 1899, p. 677. Mills and Spiller: Brain, Autumn, 1902, p. 318.

[2] Spiller: Proceedings of the Path. Soc. of Philadelphia, Jan., 1902, p. 66.

The cyst occupies almost the entire left side of the calvarium.



were found in the urine. He died with œdema of the lungs, September 14, 1901.

A large hemorrhagic cyst, measuring 15 cm. in the anteroposterior diameter, and 10 cm. in width about its middle portion, was found on the left inner surface of the cerebral dura. The brain was much compressed by this cyst, the motor area of the left cerebral hemisphere being implicated in the compressed area. Secondary degeneration, even when the method of Marchi was employed, was not distinct, as only a few black dots were found by this method in the right crossed pyramidal tract. The right hemiplegia was caused by pressure.

GLAUCOMA.

By JOHN WELSH CROSKEY, M.D.

OPHTHAMOLOGIC SURGEON TO THE PHILADELPHIA AND SAMARITAN HOSPITALS; CHAIR OF OPHTHAMOLOGY, LARYNGOLOGY, OTOLOGY, TEMPLE COLLEGE.

A report of three interesting cases of glaucoma, illustrating three fully-developed types of this much dreaded eye disease—the simple, non-inflammatory, the subacute or chronic, and the glaucoma fulminans. Once glaucoma is thoroughly established, there is very little difficulty in making a diagnosis. The stages that are mostly overlooked, especially by the general practitioner, are the early ones; and as successful treatment depends almost entirely upon the early recognition of the symptoms, it is very important to have a clear and accurate understanding of the disease, so that we shall recognize early the symptoms our patients will complain of when an eye is about to become glaucomatous.

The first case is one of the non-inflammatory type. Mrs. J., aged sixty years, married, mother of four children, a frail delicate woman. Three years before I saw her she had received a blow on the eye from a piece of wood which she was chopping, and it was from this occurrence she dated all of her trouble. She complained of no pain in either eye, nor had she ever had sore eyes, but the sight had slowly and steadily become less, until, when I saw her, she did not even have light perception. She complained, however, of subjective sensations of light. These caused her much annoyance, and were so vivid at night as to make her afraid.

The tension of both eyeballs was stony, the congestion and tortuosity of the episcleral veins were well marked. The cornea was insensitive to touch, but perfectly smooth. The conjunctiva retained its transparency, the sclerotic its pearly whiteness. The iris was of the characteristic color, and atrophic in parts, the pupil irregular in its outline and dilated, the anterior chamber was shallow. Ophthalmoscopic examination showed the lens in each eye becoming cataractous. There was a marked cupping of the optic nerve, pulsation in the retinal arteries, and the veins were large and tortuous.

CASE II.—Mrs. B., aged fifty-seven years, married, no children. Had been seized during the night with what was diagnosed as a bilious attack. This was accompanied by intense pain in the left eye, the ear, and upper teeth. The eyeball was very much inflamed, and the physician who had charge of the case used atropin, thinking that he was treating a case of iritis. After each instillation of atropin the pain increased, and became so intense that she was brought to Blockley for treatment. Upon examination there was a deep, livid injection of the ocular con-

junctiva; the cornea was cloudy, and quite insensitive to touch. In the inflamed eye she had barely light perception. The pupil was widely dilated, the anterior chamber very shallow, and the tension of the eyeball was markedly increased. Eserine was ordered and freely used, and six days after admission the pupil was sufficiently contracted to permit the performance of a good, broad iridectomy, with the result that the pain subsided within twenty-four hours, and the vision slowly returned. Ophthalmoscopic examination showed the fundus to be in fairly good condition; no cupping, but a distinct pulsation of the retinal veins.

CASE III.—Mrs. A., aged eighty-three years, married, mother of five children, none living. The following history was given: One month previous to admission she had gone to bed in perfect health. At 2 o'clock she was awakened by a most severe pain in the forehead and left eye. Before morning the eyeball became very much inflamed, and felt too big for the socket; the lids were very much swollen, and the sight was almost gone. There was some fever and persistent vomiting. By noon of the following day the right eye became similarly affected with loss of sight. Upon admission the suffering had considerably abated, the eyelids were not much swollen, but the eyeballs were of stony hardness. The ocular conjunctiva was of a dusky-red color, and extremely congested. The cornea was rough and nebulous, and entirely insensitive to touch.

The irides were discolored and the pupils dilated and oval in shape, and did not respond to light. The anterior chamber was shallow, and the vision was entirely gone. This last case illustrates glaucoma in its most terrible aspect, the course and termination being of but a few days' duration.

DEVIATION OF THE NASAL SEPTUM.

By E. B. GLEASON, M.D.

CLINICAL PROFESSOR OF OTOLOGY AT THE MEDICO-CHIRURGICAL COLLEGE; LARYNGOLOGIST OF THE PHILADELPHIA HOSPITAL; SURGEON IN CHARGE OF THE NOSE, THROAT, AND EAR DEPARTMENT OF THE NORTHERN DISPENSARY; ONE OF THE LARYNGOLOGISTS TO THE PHILADELPHIA HOSPITAL.

During my term of service in the Philadelphia Hospital several operations for deviation of the nasal septum were done at the public clinics. Of these, two were especially noteworthy because of the age of the patients. Both were old men; one of them sixty-nine years of age. Neither case complained of pain during the operation, which was done under cocaine anæsthesia, nor was there any noticeable constitutional symptoms nor any complaint of inconvenience or distress during the period while the wound was healing. Indeed, the after-treatment consisted simply of inspecting and spraying the interior of the nose. The shock of the operation, and the inconvenience of the after-treatment to the patient, might be compared to that which would be experienced from the extraction of a molar tooth.

In both cases the operation was successful, inasmuch as the nasal septa remained in the median line six months after the operations, so that adequate nasal respiration was secured, and also an aleviation of the catarrhal condition for which the operation was undertaken.

The operation performed on the two old men is the one devised by me in 1896. It consisted of a U-shaped incision around the deflected area. The vertical crura of the U are made as long as possible, in order to take advantage of the leverage which the resiliency exerted at the neck of such a quadrilateral flap has to overcome in order to spring the lower edge of the flap back into its former abnormal position.

This very important principle of leverage may be demonstrated practically by flaps of various shapes cut in the side of a rubber ball or sheet of India rubber, because the resiliency of the septum is comparable to that of India rubber. It will be seen that when a quadrilateral flap is long and narrow, that it will remain almost without sup-

port in any position into which it may be thrust by the finger; while a short, wide flap springs back into its former position as soon as the pressure of the finger is released. It will be demonstrated also that the rule that—the longer the flap the less the resiliency at its lower edge—does not apply to triangular flaps, because the width of the neck of a triangular flap increases with its length, and hence the tip or angle of even a long triangular flap cut in India rubber or septal cartilage, when bent, springs back into its former position immediately the pressure is released.

In traumatic deflections when a U-shaped incision is made about a deflected area in a septum, the original traumatism in a large proportion of cases is practically reproduced, and it is not exactly the same, because during the years that the cartilage has remained dislocated it has assumed a new shape, and hence when the deflected area is reduced or set into its normal position its resiliency tends to spring it back into its former abnormal position. However, during the years that the triangular cartilage has remained dislocated new tissue has formed which represents the so-called redundancy of deflected septa, so that when the quadrilateral flap is pushed through the septum into the median line, this redundant tissue overlaps the edges of the U-shaped incision, and serves the purpose of a splint which is adequate in rather more than 80 per cent. of cases to maintain the formerly deflected area in the median line during the healing process. During the healing process this new tissue disappears gradually, as the result of absorption and pressure neurosis; therefore, it is rarely necessary to cut away any of the redundant tissue. In practice, I cut away at the time of the operation or shortly afterward a portion of the redundant tissue only in cases where it is sufficiently great to entirely occlude the formerly unobstructed nares.

What has been said above as to the disappearance of redundant tissue also applies, to a considerable extent, to lesser and irregular deflections of the septum after the main deflected area has been brought into the median line. The condition of such areas either improves spontaneously as the result of adequate breathing space, or, if pressure symptoms persist, they are readily dealt with by removing a portion of the septum with a saw, or by using the snare or galvanocautery upon a hypertrophied turbanate. However, if necessary, trifling modification of the operation may be devised to meet unusual cases.

The technique of the operation is as follows:

A saw is introduced flatwise into the obstructed naris, and a cut is made horizontally beneath the bulging area until the saw has penetrated somewhat deeply into the bone and cartilage. The direction of the saw is somewhat rapidly changed nearly to the vertical, and the sawing continued until a gush of blood indicates that the septum has been penetrated. The tip of the saw is then thrust through the opening in the septum, and the anterior crux of the U-shaped incision rapidly made by sawing upward with the tip of the saw. However, the anterior crux of the incision is sometimes advantageously made with a knife. In fact, the whole incision might be made with a knife. The advantage of the saw is that at the base of the deviation the hard, bony, nasal processes of the superior maxillary are generally encountered, and are more easily penetrated by a saw than a knife; while the cartilage through which the anterior crux of the U-shaped incision passes is equally well cut either by a saw or a knife.

After the anterior crux and base of the U-shaped incision about the deviation has been made in the manner described, a short, probe-pointed, double-edged knife curved on the flat, is thrust through the cut in the septum from the right side (patient's left naris). The forefinger or little finger of the left hand is then introduced into the patient's right naris, so that the probe point of the knife rests on the tip of the finger. Finger and knife are now thrust backward through the nose, so that any tissues at the base of the U-shaped incision that have escaped the saw are severed until the posterior border of the deviation is reached. One edge of the knife is now turned upward, and the posterior crux of the U-shaped incision made as high up as possible upon the septum. The posterior crux of the incision is usually through bone, and it is necessary, in order to sever it, to employ lever-like movements of the knife, the tip of the finger within the left naris on which the probe point rests, acting as a fulcrum.

The U-shaped incision about the deflection has now been completed, the whole procedure occupying less than one minute. It yet remains to thrust the deflected area through the septum. If senechia exists they should be broken down with the saw, which is best done immediately before making the U-shaped incision. The forefinger (in children the little finger) of the operator is dipped into sterile water and

introduced into the patient's obstructed naris. For the left naris the right forefinger is employed; for the right naris the left forefinger. With a semi-rotary motion the finger is passed into the naris beyond the deflected area. If, as the result of defective technique, the posterior crux of the incision has passed through instead of beyond the deviation, the posterior edge of the incision is broken and dislocated into the median position. The finger-tip is then thrust beneath the quadrilateral flap into the other naris, and brought up along first the posterior and then the anterior crux, in order to ascertain that the edges of the quadrilateral flap have cleared themselves to the full extent of the incision. An effort is then made to break the neck of the flap by pressing the fingers violently upward beneath it. If there be bone in the neck of the flap, it breaks with a snap sometimes audible across a large room, and the operator may feel assured of the success of his operation: because broken bone not only remains where it is placed, but acts as a splint for the cartilaginous portion of the neck of the flap which always it is impossible to break.

This manipulation of the flap with the finger-tip is of the utmost importance, and I depend rather upon it for the success of my operations than upon the support produced by the overlapping of the edges of the quadrilateral flap, which is only adequate to prevent a reproduction of the previous condition after the most thorough bending of the flap. As for any incidental beveling of the edges of the U-shaped incision, as a means of increasing this support, it is of so little consequence as to scarcely deserve mention.

If there be any advantage in my operation over others, it is because there is only one flap to bend, and that is so situated that bone is usually encountered in the neck of the flap which can be fractured; or if the deflection is so situated as to permit, the posterior suture of the triangular cartilage may be dislocated, and the resiliency of the neck of the flap suspended during the healing process. Under such circumstances, the edge of the flap is anterior, and its crura nearly horizontal.

In the case of the two old men operated in the public clinics, after the deflected area had been brought into the median line, a tube was inserted into the formerly obstructed naris, and was worn until the next day, when it was removed and not subsequently reinserted. It is usually possible in old persons to secure a satisfactory result without the wearing of a tube during the healing process, because the most

satisfactory cases are those where the obstruction of the occluded naris is complete, because in such cases there is abundant redundant material to serve as a splint. The operation is more satisfactory in cases where the deflection extends well back upon the septum, because the neck of the quadrilateral flap then usually contains bone. For the same reason the operation is more satisfactory in mature adults than in young adults and children. The septal deviation of children are almost all "tube cases," and the wearing of a tube in a few cases in which it is necessary, causes vastly more distress and inconvenience than the original operation.

NEURASTHENIA IN CHILDREN.

By J. MADISON TAYLOR, A.B., M.D.

The disorder called "neurasthenia," occurring in children, would seem at first sight to be a rarity. The acquired form, so common in adults, secondary to a large variety of exhausting conditions, is seldom encountered among them, yet does occur with sufficient frequency to warrant attention which, indeed, it too often escapes. There are also many causes of feebleness or frailty which, in essence, are instances of "nerve tire," and are the product of injudicious or unfortunate environment or upbringing so closely allied to hereditary causes that they cannot well be separated. It was my privilege in the article in the *International Med. Mag.* for August, 1896, to call attention to the importance of recognizing these cases and pointing out the measures by which improvement can be secured. A certain proportion of the hereditary neurasthenics will remain such throughout life, in spite of the best opportunities and influences. These are often observed among families of wealth and liberal conditions, where no blame can be attached to parents for lack of efforts to repair inherent defects. A much larger number of children of obvious feebleness, the product of developmental defects and faulty hygiene, are capable of satisfactory improvement. The most difficult problem is for the medical adviser to obtain control of the case for a sufficient length of time. The next difficulty, and one which must be thoughtfully met, is to secure the right kind of caretaker.

The phenomena of neurasthenia in children are obvious enough if rightly interpreted, and are relatively free from the confusing factors common in adults, the outcome of bad habits, hysteria, and mendacity. A child who is described as "so fond of reading," the inference being that it thereby exhibits a thirst for knowledge is, in nine cases out of ten, lacking in central vigor or is simply lazy. No child of healthy impulses or physique will be content to idle its time poring over books, but will take advantage of, or make opportunities for, the investigation of natural phenomena, or give evidence of spontaneous impulses toward

activities. Upon scrutiny, such children will be found lacking in organic vigor or integrity, and will exhibit defects of development, induced by insufficient use of its locomotive or other machinery. The child may not be then neurasthenic, but its central nervous system readily suffers from the effects of disease, and the symptoms of neurasthenia follow. The one thing lacking may be a normal direction of impulse to act in such a way that symmetrical powers can be developed. Again, it may be observed that a child is hypersensitive to external impressions, to such a degree that action and reaction follow so swiftly as to exhaust power and prevent development, thus inducing a state of lowered energizing, from which it may never fully recover. It is true that fairly large capacities for neural energizing are compatible with enfeebled limbs and trunks. It is also true that where the muscles and brain are equally developed, a much more efficient citizen will be produced than if endowed with mental activity in excess of physical. Again, it is not rare to find, in earliest childhood, evidences of hypertension, both neural and physical, which induce an over-strenuousness, and this excess of impulse, even in the avenues of right living, is followed by speedy exhaustion. Some such children rarely or never are adequately relaxed, so that they fail to get enough rest one day to fit them for their play or work the next. If they survive, it is a question if they can, or do, fulfill reasonable expectations, warranted by their abilities. As adults these folk may accomplish much for a brief space, but fall easy victims later to overwork; or they drop back in the race for success from first to middle place, like an over-ambitious horse, who, on a long journey, rushes his hills and fails to take his ease on down grades. Exhaustion inevitably follows in the child or adult who puts forth needless effort to accomplish each item of work, or who, when the opportunity comes to relax, or to step aside for a better start, comes to the effort with muscles or nerves in no condtion for normal action, but exhausted by too prolonged "expectant attention."

The phenomena of hypertension need to be differentiated from those of hypotension. In the first we have greater physiological irritability, indicating measures to allay this in the many states which obviously follow. It is well to use little fluid at meals, and yet hypodermoclysis is of peculiar value. For such, surface massage, deliberately administered, is helpful, followed by slow, active movements and deep breath-

ings. In conditions of hypotension a rather different set of phenomena will be encountered, weak yet irritable pulse, apathetic states, etc. For such, much rest is required, forced feeding, possibly alcoholic beverages, stimulating baths, salt douches, etc., oxygen inhalation, deep massage, followed early by resisting exercises.

In estimating the causes of neurasthenia, Kraepelin, in a recent paper, points out the harm which may result from a failure to differentiate the neurasthenic state from certain defined nervous affections, the treatment for which should be in many instances distinctly different. What holds good in the adult cases also obtains in respect to neurasthenia in children. These conditions are dementia precox, neuroses, due to over-worry, fatigue, or phobias, maniacal, depressive, or circular insanities, and certain progressive paralyses. The milder forms of circular insanity are peculiarly liable to simulate neurasthenia. The emotional depression seen in psychoses, the absence of volitional power, and the rapid development of symptoms should make a diagnosis plain. Although rare, suicidal tendencies are met in children. As infancy is left and youth approaches, many psychic conditions can be recognized, closely resembling those of adults, and Allbutt has recently announced a view of neurasthenia that it is an individual weakness in which exhaustion takes place in excess of repair with a lowering of nervous potential, and imperfect recovery from depressing states. Hysteria is a psychosis not expressed by disorders of intellect, but in defects of character and emotional disturbances. Its real nature is hidden by simulated physical disorders whose basis is interference with normal inhibition. Marked depression of spirits, if primary and persistent, is more characteristic of melancholia or hypochondriasis. This last is not a step between neurasthenia and insanity, nor is it insanity, though closely allied to it. It consists in a fixed idea which nearly approaches a delusion, but does not cross the narrow line as often as might be expected. Neurasthenia with obscessions is nearer to insanity, especially where there are impulses to do a wrong thing, or to satisfy a morbid desire. In neurasthenia a lowered capacity to maintain attention during times of depression is characteristic, but distinct lapses or breaches of memory indicate hysteria or some definite diseased mental process. At all times it is necessary to give close study to the diagnosis of neurasthenia, bearing in mind that it may be merely a condition superadded to some more definitely diseased state

which is made much more serious by this impairment of reparative power.

In dealing with the complex problem thus presented, the physician must always bear in mind the practical importance of keeping the psychic factor in view. His mental influence rightly used in the direction of wholesome, dominant suggestion is just as efficient as with adults; possibly more so. He must be permitted to know his patient, and to deliberately win confidence and exercise volitional control. He must be permitted full opportunity and enjoy the co-operation of the family. If not, the character of the conditions, their double quality of physical and psychic syndrome offers grave dangers in the formation of bad mental habits which may lay the foundation for future hysteria or hypochondriasis.

It is recognized by American oculists that a frequent cause of neurasthenia and denutrition is to be found in eye-strain.

In the study of the child who exhibits evidences of neurasthenia, certain organs obviously need attention. The heart in early life is subject to much disturbance of its nervous mechanism; cellular stability is still far from established and in no organ is this more constantly exhibited than in the cardiac cycle. As is well known, the pulse of an infant varies widely under many normal reflex perturbations. Heitler has pointed out the reflex exaltation of the pulse to be common in neurasthenics. The cardiac dullness is found to alternate with the liver dullness. Under many slight irritations as the pulse increases in volume the cardiac dullness lessens and the liver dullness increases. Cardiac neuroses are common in children. Murmurs must be carefully estimated on this basis, and too much significance should not be allowed to alterations in the phenomena learned by percussion, rhythm changes, or data acquired through auscultation. The lungs are liable to exhibit many phenomena requiring careful interpretation. Dyspnœa arises under various conditions. Sometimes the breathing is hurried by the simplest, often apparently insufficient, causes. At others, much exertion is surprisingly endured.

Probably the best tonic in the whole category is air. This should be made to enter the lungs freely and constantly. No other agent can accomplish so much in enhancing organic competence as the exercise of full pulmonary expansion. I am prepared to promise better results through opportunities to teach proper breathing movements to

the frail child than from all the other means at our command. Take two cases, similarly conditioned, and let one enjoy all the privileges of the best environment purchasable, food, country air, horses, mountains or seashore in summer and subtropical climates in winter, and yet permitted to dawdle according to the taste or dictates of a mind defective in healthy impulses, trying to gain strength by all such slow measures. Contrast the progress of such an one with another who shall enjoy only fairly good surroundings, and yet have the personal guidance of an expert in passive and active exercises and respiratory gymnastics, and much more swift and satisfactory results will follow.

The perpetual formation of poisons in the blood exert their baneful effect upon the eliminating organs in proportion as these are, or are not, supplied with adequate oxygen by which to destroy them. Hence the kidneys must be ascertained to be functionally competent, or to be made so, if practicable. Studies of urinalyses too often fall short of the points which will elucidate most.

Here also comes up the large and greatly increasing subject of the physiology of the ductless glands which is destined to outweigh all other studies in organic changes in the near future. When we learn the practical lesson of the full significance of the minute secretions of these ductless glands and can control their output, and add to this a knowledge of their arterio-motor mechanism, we shall be in possession of the key to the progress of most disease processes. Already much is known of practical value, but few medical practitioners have a working knowledge of the facts at hand. We ought soon to be able to recognize gross changes in developmental steps in functional activities of the glands and at least modify them. Whether this shall be by the use of the secretions themselves or by exerting control of the arterio-motor mechanism by which changes can be lessened, I do not know. We must hold the matter constantly before our attention and fortify ourselves by keeping abreast of the advances in organotherapy. Meanwhile, we cannot neglect the use and empirical knowledge of hygiene, etc., now in our possession.

TREATMENT OF THE NEURASTHENIC CHILD.

I may be permitted to use a modified quotation here from my article above referred to:

The upbuilding and repair of all children, especially those who are

weakly or neurasthenic, should be considered on broad principles, the basis of which is elaborate thoroughness and abundance of time. This involves special attention to dietetics, including a critical estimation of varying states and capacities of digestion, well chosen hygienic measures, and the hopeful use of some drugs. There must be insisted on for such, both during average health and during illness and convalescence, more rest for the mind and body than is necessary for the average child. All outings and exercises, both active and passive, should be supplemented by rest, lying down for as long a time, it may be minute for minute, as the active periods. The rest is necessary to enable lowered organic processes to regain their customary tone, and especially to secure definite gains. It will be often necessary to precede food by a period of rest, to enable the digestive activities to have full play; otherwise the highly sensitive nervous distribution to the digestive apparatus will fail of its full energizing. Mental or emotional agitation impairs the even flow of the circulation, so necessary for the best working of weakened organs, particularly the brain, whence governing impulses perpetually flow, dominating the body and spirit. Therefore, too, the emotions must in the weakly be not only kept well under control, but subjected to the least possible disturbance or exaltation. The temperaments (or mental attitudes from which they view life) of all children require steady and patient training. Even in the home a full recognition of this is needed. In the case of strong children, equipped with clear, dominant, healthy minds, it is undoubtedly true that fair results come somehow from very diverse and ill-directed influences; but for the weaker ones, impressionable or apathetic, is required a thorough, conscientious study and specially directed measures.

For such little folk it is not enough to prescribe suitable medicines and enumerate casually a list of easily digested foods which the mother shall provide, nor to direct proper bathings, outings, and other general measures. A thorough systematization of the entire daily life of the child is infinitely more efficacious than the most accurately selected medicines or the use of that inumerable host of children's foods with which, in the form of specious descriptive circulars, the enterprising chemists flood our morning mails. The best tonic for the stomach is food carefully prepared and served, such as a fairly intelligent mother in even the humblest walks of life, if rightly directed,

can readily afford, but always provided that the preparation, seasoning, the times and circumstances of administration be wisely chosen.

Predigestion of food-stuffs offers undeniable safeguards to the weakened, toneless, digestive tract, but robs the pabulum too often of that savoriness which is essential to acceptability, and hence imperils appetite.

While exercising care as to quality and preparation of foods for weakly or neurasthenic children, it is imperative to bear in mind the need for suitable variety. A child will often be presented who is fed with the utmost care and regularity, oftentimes under the best of medical advice, and yet its progress comes to a standstill or it obviously retrogrades. Upon inquiry there will be revealed much sameness in the diet list, otherwise properly adjusted to the condition for which it was originally outlined. The little victim's soul comes to loathe and abhor the sight of flabby paps, occurring in dismal routine, or the same old wearying round of bread, meat, and a dab of vegetables. If to these is now added a more varied dietary, revising the *menu* day by day, even lapsing into a taste now and again, of articles ordinarily forbidden, yet savory and tempting, great progress will soon be obvious.

The points which too often do not obtain adequate attention are the thorough systematization of the when, where, and how much of these foods shall be taken; what varieties shall be insisted upon; the times, kind, and suitability of bath; the amount and character of exercise both passive and active, and, above all, definite periods of rest before and after feeding, so that the organs shall be able to act deliberately. First, then, when confronted with an ailing child, one who is not ill, but far from well, when appetite is variable but small, when sleep is restless, the digestive organs manifestly disturbed and temper fretful, a child who fails to hold its own in play among its fellows, and what cannot be ignored, whose weekly school report shows decided backsliding—first, look the little fellow over thoroughly and search all organs for sources of reflex irritative strain.

There may not be one organ more amiss than another, though the most obvious results will usually be seen in that avenue to all vital power, the *prima via*. There may be yet no falling off in weight (a far more instructive index in a child than in an adult), nor an obvious anæmia. There may be a quicker pulse than ordinarily; a change in the heart-sounds which the initiated will recognize, but cannot so

clearly describe; there probably will be found, if so much trouble is taken, a rise in temperature, slight but unmistakable at certain times, at others subnormality is well marked, and there is great probability that neither the attention nor other exertion is readily sustained. The child, in marked contrast to its healthy comrades and itself at other times, is willing to sit aimlessly, if not made a martyr by energetic task-masters or by an over-strenuous conscience which drives its willing victim to the verge of exhaustion, and often over it into the pit of complete collapse.

Such cases as here pictured are common enough, if only the eyes are open to see them. They escape attention only too readily until some malady seizes them in all their pitiable weakness, and life is speedily quenched. It is a worthy quest, then, to seek out and rescue these from, it may be, no picturesque fate, but an ever-present menace; to rehabilitate these unresilient little bodies, and even make them better than before; to put them in the way of getting a sound bodily equipment for their life-work anon.

Here is a sketch of modified rest treatment which produces excellent results when all other efforts have failed to start a child along the line of progress: Put the little one in bed from a few days to a week or more, and write down distinctly for the mother a strict schedule, giving the exact hours for feeding and bathing, etc. These may be the ordinary three meals, with some little fluid food taken between times, or, better, direct four meals to be given in the day, at, say, seven, twelve, four, and eight o'clock; the largest meal at noon. Omit the drugs hitherto given and add digestive ferments, or malt, or both. Let the day begin with a sponge-bath in a warm room; then a light breakfast daintily served. In the early afternoon let some one rub into the trunk and limbs an oil; olive oil will do, and much of it is thus absorbed, especially if one-third part soap liniment is added, which probably aids the osmotic action. Lanolin, diluted, is best of all; changes are desirable in all skin applications. This serves as a form of passive exercise and also as a nutriment, or at least as a tonic to the skin, circulation, and cutaneous nerves. The surface should be thoroughly wiped off afterward, that no foulness remain. Above all, in the early course of these measures, if the child manifests a desire for toys, they may be allowed sparingly, but aggressive entertainment by officious persons is a harm and an offence and should be strictly

forbidden. After a few days or weeks the range of one sunny room may be permitted, but still the child should be let alone, and in most cases it will be happy enough and amuse itself.

Frail children require systematic development of their various organs, as well as of their muscles. To be sure, it seems scarcely practicable to increase the power of some organs, as the stomach or kidney; nevertheless it is possible to do so. It is abundantly obvious that the heart, the eyes, and the lungs and the skin can be developed, and it is equally important that all these organs should receive attention in the aggregate and separately, especially where there is a manifest underdevelopment of the one or the other, which then should receive specific attention.

THE ASSOCIATION OF VALVULAR HEART DISEASE WITH TABES.

By B. FRANKLIN STAHL, M.D., B.S.

LECTURER ON DIETETICS OF THE SICK AND INSTUCTOR IN PHYSICAL DIAGNOSIS, UNIVERSITY OF PENNSYLVANIA; VISITING PHYSICIAN TO ST. AGNES' HOSPITAL; MEDICAL REGISTRAR, PHILADELPHIA HOSPITAL.

The occurrence of organic heart disease in association with tabes has been referred to frequently, and the cases studied in this paper would seem to justify the varying conclusions that have been reached by writers upon this subject.

That the association of the two conditions is not regarded as of common occurrence or of great importance is evidenced by the failure of certain American authors to recognize endocardial involvement as a complicating factor in cases of tabes. On the other hand, literature on this subject records so many instances of circulatory involvement as a feature of the disease that it would seem to justify a more extensive notice of this complication, and more emphasis of its likelihood to demand consideration in the treatment of these cases.

The cases examined in the nervous wards of the Philadelphia Hospital showed a marked involvement, not only of the valves of the heart, but also pronounced changes in the arteries and presumably in the entire circulatory mechanism. It has not been difficult to find patients of twenty-one or thirty years of age with radial arteries as rigid as should be the vessels of men who have lived their three score and ten years.

The frequency with which valvular disease is reported in cases of tabes varies materially in the findings of different examiners. It is a question if the personal equation has not influenced these results. They may have been further affected by the diversity of opinion as to what modification of the heart-sounds indicates the existence of valvular disease. The post-mortem records herewith submitted lend emphasis to the now generally accepted fact that the presence of murmurs does not invariably indicate structural change of the valves.

In my report of cases I have taken the precaution to have the diagnosis of the heart condition verified by a colleague.

Frequency.—Marie observes that in every four or five cases of sufficiently advanced tabes, one more or less well-marked case of cardiac change will occur.

Jaubert reports thirty-seven cases of locomotor ataxia associated with cardiac lesion. Twenty of these cases presented valvular lesions, and the remainder showed myocardial or aortic change. In contrast with this is the report of the examination of fifty-six cases by Balacakis, who found three instances of valvular disease.

Grasset has reported twenty-four cases of tabes showing various cardiac lesions, while Berger and Rosenbach have reported seven cases of the disease in which there was aortic insufficiency. Five of these cases were women, and two men.

Angel reports a series of twelve cases, in three of which there was developed a diastolic murmur after prolonged effort, and which disappeared after a night's rest.

Gutman says that in a hundred cases of tabes he found only three cases of valvular heart disease.

Groedel has examined 153 cases, and found two cases of aortic insufficiency, one of mitral insufficiency, and one of mitral stenosis.

Friedrich Enslin (*Deutsche Med. Wochenschrift*, April 21, 1898), in an inaugural dissertation on the occurrence of aortic disease in association with tabes, reports from the Moabiter Hospital seventeen cases, of which number seven exhibited aortic insufficiency, seven had endocarditis, and three had aneurisms.

Vulpin and Charcot have referred to the frequent association of tabes and cardiac disease.

Dr. Pearce Bailey roports the examination of eleven cases, all of which were in the earlier stages and able to walk to a clinic, and in none of the group were any murmurs discovered. In another series, of advanced cases, of men past middle life, he was able to discover only one case having cardiac valvular disease. This man was an exception to the rule that the cases were past middle life, he being thirty-three years old, and he was both syphilitic and alcoholic, and had aortic insufficiency.

Causation.—It is an open question whether there is a direct causal relationship between the two diseases. There is a considerable diver-

sity of opinion upon this subject among writers. Leyden regards the coincidence of the diseases as accidental; Berger and Rosenbach do not express an opinion regarding its causation; while Gowers and Oppenheim have expressed the opinion that where the two diseases are associated it is the result of the same cause, namely, syphilis. It would help to determine this point if a sufficient number of cases could be studied to discover whether valvular heart disease existed before the tabes had advanced to the extent of exercising an influence in its causation.

Aortic insufficiency is said to be the most frequent form of valvular disease found in these cases. Indeed, some authors report that they have not seen involvement of any other valves. This is true of the cases reported by Berger and Rosenbach.

Sex.—There is a preponderance of the male sex in the cases reported, except those cited by Berger and Rosenbach, where five of the seven cases were females.

The following cases were examined by me in November, 1898, and comprised all the cases of tabes in the nervous wards of the Philadelphia Hospital at that time. Since then eleven cases have died, and the post-mortem findings in seven of the cases are appended:

Case.	Age.	Aortic Area.	Mitral Area.	Radial Artery.	Pulse.	Specific History	Urine.	Duration.	Eyes.
1	35	2, sound blurred.	2, sound accentuated.	Corded.	98	No.	Albumin casts.	7 yrs.	Normal.
2	58	Both sounds very distant; 2, sound scarcely heard.	1, sound murmurish.	Corded.	94	Denied.	Negative.	1 yr.	Pupils very small and unequal.
3	48	1, sound distinct; 2, sound very feeble.	1, sound blurred.	Corded.	96	No.	Transient albuminuria; no casts.	6 yrs.	Pupils very small and unequal.
4	53	1, sound weak; 2, sound normal.	1, sound weak; 2, sound accentuated.	Normal.	72	Yes.	Negative.	6 yrs.	Pupils unequal.
5	59	Faint systolic murmur.	Loud systolic murmur.	Somewhat rigid.	62	Yes.	No report.	12 yrs.	Pupils pin-point.
6	36	Systolic murmur.	Systolic murmur.	Corded.	104	Yes.	No report.	1 yr.	Normal.
7	63	2, sound blurred.	Normal.	Rigid.	98	No.	Albumin casts.	12 yrs.	Marked arcus.
8	81	Both sounds distant and feeble; second sound very feeble.	Normal.	Normal.	96	Doubtful.	No report.	Not known.	Marked arcus.
9	50	Loud systolic murmur.	Systolic murmur.	Rather rigid.	88 Irregular.	Yes.	Negative.	6 yrs.	No arcus.
10	40	Double murmur.	Presystolic (with thrill) murmur; sounds booming.	Corded.	104	Yes.	Negative.	6 yrs.	No arcus.
11	50	Systolic murmur; 2, sound weak.	Presystolic murmur, with thrill.	Corded.	108	Yes.	Albumin.	3 yrs.	Pupils unequal and irregular.
12	57	Sounds distant; 2, sound blurred.	1, sound murmurish; both sounds blurred.	Rigid.	100	No.	Albumin; no casts.	20 yrs.	Pupils unequal.
13	60	Faint diastolic murmur.	1, sound blurred.	Rather corded.	94	Yes.	Albumin.	16 yrs.	No arcus.
14	21	Both sounds accentuated.	Systolic murmur.	Corded.	88	Doubtful.	Albumin.	1 yr.	Blind.
15	60	2, sound blurred.	Normal.	Rigid.	76 Intermits.	Yes.	Negative.	5 yrs.	Pupils unequal.
16	62	Diastolic murmur.	1, sound blurred.	Somewhat corded.	104 Intermits.	Doubtful.	Negative.	4 yrs.	No arcus.
17	48	Normal.	Both sounds booming.	Corded.	106	Yes.	Negative.	6 yrs.	No arcus.
18	45	Normal.	Both sounds booming; apex displaced to right.	Somewhat corded.	98	Denies.	Trace albumin.	9 yrs.	No arcus.
19	56	Normal.	Presystolic murmur (with thrill).	Rigid.	90	Denies.	Negative.	10 yrs.	No arcus.
20	48	Sounds scarcely heard.	Systolic murmur.	Corded.	89	Yes.	Negative.	7 yrs.	No arcus.
21	35	Normal.	Normal.	Normal.	80	No.	Negative.	10 yrs.	No arcus.
22	60	Double murmur (after exertion).	Double murmur.	Rigid.	94 Irregular.	Yes.	Negative.	6 yrs.	Arcus.
23	54	Systolic murmur.	Systolic murmur.	Somewhat corded.	84	Yes.	Negative.	5 yrs.	Pupils unequal.
24	53	Normal.	Normal.	Normal.	70	Denies.	No report.	5 yrs.	Pupils unequal, very small.

SUMMARY.—In this series it will be noticed that the heart-sounds at the mitral area were normal in four cases; while at the aortic area they were normal in five cases.

A mitral systolic murmur occurred in six cases.

A mitral presystolic murmur occurred in three cases.

A double mitral murmur occurred in one case.

An aortic systolic murmur occurred in five cases.

An aortic diastolic murmur occurred in two cases.

An aortic double murmur occurred in two cases.

The radial artery was appreciably affected in all but three cases.

The pulse averaged more than ninety in a majority of the cases.

The presence of arcus has been noted because of its relation to senile vascular changes. This sign was present in three cases.

In this group of cases one female is reported.

Age.—The disease occurred in nineteen of these cases during the fourth, fifth, and sixth decades.

POST-MORTEM FINDINGS IN CASES REPORTED.

CASE IV.—Pericardial cavity and heart are normal.

CASE XII.—Pericardium contains about 150 c.c. of slightly clouded straw-colored fluid, with a few flakes of fibrin. Parietal layer greatly thickened. Serous surface covered with a trabeculated adherent layer of blood-stained fibrin; beneath the fibrin are numerous minute hemorrhages. Visceral layer entirely covered by an adherent layer of shaggy fibrin, which in places appears as rugæ. Fibrin somewhat blood-stained, but not to same extent as that on the parietal layer. The accumulation of fibrin is most marked along the right border at the base, and to a less degree along the left border. Heart not opened; saved for gross specimen.

CASE XIII.—Heart: a few milk spots on visceral layer of pericardium. On opening the heart the right ventricle and right auricle contained chicken-fat-clot. Heart muscle is fairly firm and shows no marked gross changes. Coronary arteries are slightly sclerosed. Aortic leaflets show few striations. Mitral valve shows decided thickening along its free border; valves on right side of heart show no gross lesions. Arch of the aorta shows marked atheroma. Aorta just above the aortic valves measures 8½ cm. in circumference. Pericardium is surrounded by very little fat. Sac contains about 5 cm. of a clear, though colored, fluid.

CASE XVI.—Pericardium, normal. Heart, normal. Aorta is the seat of extensive atheroma and calcifications.

CASE XVIII.—Pericardium in relation to right auricle was thickened. The heart presented a large milk spot over the right auricle, otherwise nothing of interest. This man died on surgical from cellulitis of face and neck, and chronic pleurisy.

CASE XX.—Pericardial cavity, negative. Heart, myocardium pale yellowish red in color and slightly decreased in consistency. Aortic valves reveal a few patches of thickening and calcification, otherwise heart negative.

CASE XXII.—Pericardium contains a moderately increased amount of fluid. Both layers of the pericardium are smooth and glistening. Right heart contains currant-jelly-clots and is relapsed. Left ventricle contracted and contains small clots. Valves of right side are of normal aperture, and valves themselves are smooth. Mitral orifice admits two fingers; its anterior leaflet shows spots of atheromatous change. The aortic aperture measures 8 cm. The leaflets are calcified at their attached margin, and the copara aurantia are large. The lunula on two of the leaflets are fairly well retained, on the third there are some small warty excrescences immediately above as well as in the sinuses of Valsalva.

CASE XXIII.—The aorta presented marked atheromatous changes of calcification. The sinuses are considerably dilated; walls of coronary arteries are slightly thickened. Heart muscle is of brownish-red color; left ventricle, 20 mm. thick; right ventricle, 6 mm. thick.

THE UNILATERAL OCCURRENCE OF KERNIG'S SIGN AS A SYMPTOM OF FOCAL BRAIN DISEASE.

By JOSEPH SAILER, M.D.,
MEDICAL REGISTRAR TO THE PHILADELPHIA HOSPITAL.

I desire to report two cases in which Kernig's sign was present only on one side, and appeared to bear some reference to a cerebral lesion on the other side of the brain. This sign was described by Kernig in 1883 before the Medical Society of St. Petersburg, and the next year published in German. His attention was first directed to the phenomenon in a patient recovering from epidemic cerebrospinal meningitis. This patient could walk perfectly well, could lie in bed with legs extended, but whenever she sat in a chair she found it impossible to extend the legs on the thigh beyond a right angle. Subsequently he studied fifteen cases of meningitis, nine of which were confirmed by autopsy, with reference to this sign, and found it present in all. It could be elicited whether the patient sat up, lay on the back or on the side. He describes it as a flexion contracture in the legs (and occasionally in the arms) when the thigh is flexed to a right angle upon the trunk. Under these circumstances any attempt to extend the leg on the thigh meets with severe resistance as a result of contraction of the hamstring muscles, and it is impossible to extend the leg beyond an angle of 135°, or even, in extreme cases, beyond a right angle. When the thigh is extended the hamstring tendons are relaxed and soft; when, however, the thigh is at a right angle to the trunk and an attempt is made to extend the leg they become tense and prominent. The contraction is not ordinarily associated with pain, nor with any increased rigidity in any other part of the body, and Kernig particularly noted that the retraction of the head did not become greater when the patient sat up. He states that the sign usually persists long into convalescence; it may vary from time to time in the course of the disease; it is not produced by mechanical irritation of the sciatic nerves, and may, as he noted in his original communication, occur in certain other conditions, although in all of those that he observed there was reason to believe that irritation of the membranes existed.

The literature on the subject is very inconsiderable, although the presence or absence of the sign is now usually mentioned in connection with a suspected case of meningitis. The first important article on this subject was that of Friis, who found it present in 74 of 86 cases, and was not able to exclude it positively in all of the other 12. Henoch also obtained it frequently in children suffering from meningitis, and Blumm mentions its existence in 7 of 9 cases. In one case the contraction of the muscle came on suddenly and was painful. Bull obtained it in all positions in 2 cases: one a tumor of the cerebellum, and the other a thrombosis of the left lateral sinus. Netter, in an extensive series of investigations which practically called general attention to the existence of the sign, found it present in nearly all cases of meningitis. The attention of the American profession was directed to this subject by a paper by Herrick, read before the American Medical Association. He found it present in 17 of 19 cases of meningitis and only twice in 100 other cases, one of which was a case of subdural hemorrhage, and one a case of gonorrhœal rheumatism, as a result of which the patient had lain with the knees flexed for a period of four weeks. He failed to observe it in cerebral hemorrhage, brain tumor, and other intracranial conditions. In other cases the absence of the sign was of great value in excluding meningitis. He called renewed attention to the fact that the sign can be elicited if the patient either sits or lies down. Its presence has also been reported by Widal and Merklen in a case in which the lesion was found to be a clot pressing upon the anterior surface of the pons and medulla, and by Thyne in a case in which there was hemorrhage into the left lateral lobe of the cerebellum, and an effusion of blood over the right occipital lobe and into the fourth ventricle. The patient also had retraction of the head, but meningitis was not present. The sign is more often absent in tuberculous than in any other form of meningitis. Henoch was the first to mention this fact. Netter, Herrick, and Dieulafoy have all confirmed it, and Packard has reported 3 cases of tuberculous meningitis in children aged fifteen, sixteen, and four months, in which the sign was persistently absent. With the exception of one of Herrick's cases, in which the sign was elicited in the unaffected leg of a woman suffering with gonorrhœal gonitis, mention has not been made of its unilateral occurrence.

During the past year I have had under my care in the wards of the

Philadelphia Hospital, two cases in which the sign was unilateral and appeared to be a symptom of focal encephalitis. The cases are intrinsically interesting, but possess no common points except this peculiar phenomenon.

CASE I.—The patient, W. H. P., a white man, aged twenty years, laborer by occupation, was admitted to the Philadelphia Hospital on September 16, 1901. He was partially delirious, but could answer simple questions. He complained of severe headache and pains all over the body. The temperature on admission was 104°; there was slight abdominal tenderness and an occasional dry cough. His family history was indefinite; his mother had died of Bright's disease; one sister was dead; his remaining relatives were living and well. His previous history was obtained from his father, who stated that he had had whooping-cough, chicken-pox, measles, and malaria in childhood.

In 1888 he had a moderately severe attack of typhoid fever, and at Christmas, 1899, an attack of what was diagnosed as spotted fever, which lasted for about three weeks. This came on suddenly, and it was necessary to bring him home in a patrol wagon. There was a double history of an attack of appendicitis about a year ago, for which no operation was performed. For four or five weeks before his present illness he had been working on an ice-wagon. The history of his present condition was as follows: For about two weeks he had been complaining of pain in the side, backache, and headache. On September 14th he came home from work complaining of intense headache, and soon became delirious; he was not able to sleep, and on the 15th became violent. He had a serous diarrhœa, with watery, yellowish stools, and seemed to be very feverish. Upon admission the following notes were made: A well-developed, well-nourished male; numerous tattoo marks on both arms; pupils equal, react to light and accommodation; tongue heavily coated, protruded in a straight line without tremor. Pulse was strong, regular, of good volume and good strength; chest was well formed, expanded equally and well. The abdomen was negative, with the exception of slight tenderness in the right iliac region. No nervous symptoms were noted on this day. On the following day, however, when I first saw the patient, he was acutely delirious. The head was slightly retracted, although the muscles of the neck were not hard. The pulse was of medium volume. The left arm was strongly flexed and distinctly rigid. The right arm was normal. The lungs were normal; the cardiac dulness commenced at the third interspace and extended laterally from the mid-sternum to the left parasternal line; there was a weak, diffuse apex-beat in the fourth interspace. Auscultation revealed gallop-rhythm, and a slightly accentuated pulmonic second sound. The liver extended from the sixth rib by the costal border; the spleen was not palpable, but was slightly enlarged to percussion. The stomach, examined by auscultatory percussion, did not extend below the costal border; there was no eruption on the skin; the inguinal and post-cervical glands were small but palpable. There was slight hyperæsthesia over the abdomen. The supra-orbital nerves and the suprabulbar fossæ were not tender, and the patient did not manifest any discomfort upon strong percussion of the skull. The reflexes were not exaggerated. Kernig's sign was readily elicited in the left leg. It was possible to extend the right leg in an almost straight line with the thigh *when the latter* was flexed at right angles upon the abdomen. Posteriorly nothing

abnormal was noted aside from a slight increase in vocal resonance at the base of the left lung, and numerous subcrepitant râles at both bases. A provisional diagnosis was made of tubercular meningitis, associated with hypostatic congestion of the base of the left lung, and with acute nephritis. On September 18th the patient was distinctly worse; he had been delirious all night, and slept only for short intervals; he complained of pain in the precordial region; there were still signs of hypostatic congestion of the lower lobe of the left lung posteriorly. When roused there was distinct lagging of the left upper eyelid. The mouth was drawn toward the right side, and the right side of the face was wrinkled, while the left side was smooth. The reflexes were not changed. The following day the head was turned to the left side; the left pupil was smaller than the right and seemed sluggish. The tongue was dry and rough and protruded to the left side. There was marked flexor spasm in the left arm, which seemed to be paretic, at any rate the patient did not use it to remove irritants. The right arm was moved freely and appeared to be normal. The knee-jerks were slightly increased, especially on the left side, and Kernig's sign was distinctly present in the left leg, which in addition was slightly paretic. The patient now had incontinence of urine. On September 20th the condition was about the same; the head was turned strongly to the left and could not be forcibly turned in the other direction. The left arm was firmly flexed at the elbow, and resisted straightening. The right leg was not moved voluntarily; in the left leg occasional irregular movements were noted. Kernig's and Babinski's signs were both present on the left side but not on the right. The apex-beat was in the fourth interspace, in the nipple line. A systolic murmur was heard on this day for the first time, most distinctly at the apex, and there was moderate accentuation of the second pulmonic sound at the base. At midnight of this day the patient had a hemorrhagic eruption over the entire body, consisting of very slightly raised, small round spots that did not disappear upon pressure. There were also hemorrhages under the conjunctivæ, and in the mucous membranes of the lips and mouth. The patient could not be roused, and it is possible that he was deaf on both sides. The head was retracted and rotated to the left; the right pupil was widely dilated and reacted promptly to light; the left was contracted and reacted very slowly; there was chronic drooping of the left eyelid. Respiration was of the typical Cheyne-Stokes variety. The pulse showed a very remarkable alteration, it was full, rapid, and receded quickly—a typical water-hammer pulse. Upon auscultation of the heart a loud systolic murmur could be heard at the apex and at the pulmonic cartilage; there was also accentuation of the second pulmonic sound. The left arm was flexed over the chest and could not be extended by force; the right foot was not moved even when the sole was vigorously irritated. There was marked ankle clonus on both sides. The knee-jerks were increased, more on the left side. Kernig's sign and the Babinski phenomenon were both present only on the left side. The patient was entirely unconscious and only groaned occasionally. On September 21st he grew gradually weaker, the pulse becoming smaller; respirations, however, were slightly more regular; the reflexes remained increased until death, which occurred at 6.40 A.M.

During the course of the case the urine was examined on the 17th and found to contain albumin and numerous granular and blood casts. The specific gravity was 1014. On the 17th the leucocytes were 17,600; on the 19th, 27,000. The Diazo reaction was tested on the 18th and was not present. Blood was taken for the Widal reaction on the 18th, and the report returned from the City Laboratory on

the 20th that it was not present. On the 20th, Dr. Pfahler made a spinal puncture and also withdrew some blood from the median basilic vein of the right arm, under strict aseptic precautions. Agar tubes were inoculated with this material, and on all of them cultures of a staphylococcus that became golden-yellow appeared. These cultures were pure. On the 21st the autopsy was made by Professor Coplin, who has kindly permitted me to abstract his notes. Rigor mortis was unusually pronounced; the pupils unequally dilated, the left more than the right. The whole body was covered by a petechial eruption which involved all the visible mucous membranes. Upon opening the body hemorrhages were found in all the serous membranes. The right side of the heart was slightly dilated; the tricuspid, pulmonic, and mitral valves were normal. The anterior and left aortic leaflets were partially destroyed, and the edges showed recent inflammatory vegetations. Upon the remaining leaflet only the ventricular surface was involved, and it was slightly rough and contained a few small vegetations. The left lung contained an infarct upon the lower edge of the upper lobe; the lower lobe showed intense hypostatic congestion. The spleen was enlarged and soft; the kidneys showed cloudy swelling; the meninges were congested, and there was a softened area in the motor region on the right side. The right tibia was slightly deformed, and upon incision was found to be the seat of an old osteomyelitis, the marrow having been largely converted into compact bone, and the compact substance being unusually dense.

The autopsy, therefore, confirmed the final diagnosis as far as the heart, kidneys, and lung were concerned. We had supposed during life that a meningitis existed, and as a matter of fact the membranes were intensely congested and œdematous, and over a small area of the ascending frontal gyrus at the level of the second frontal convolution they were slightly altered. The main lesion in the brain, as subsequent examination showed, consisted of an area of hemorrhagic softening in the middle of the ascending parietal convolution, and extending inward through the centrum ovale toward the internal capsule, which, however, it did not involve. Surrounding this there was an area a few millimetres wide, of partial discoloration of the brain, apparently due to œdema. At the autopsy Dr. Coplin permitted me to make cultures from the brain and gall-bladder, and from both an organism was obtained which was similar in all respects to the one obtained by Dr. Pfahler in his cultures from the blood and cerebrospinal fluid. This organism has twice been injected into rabbits, once intravenously, without producing fatal effects. Dr. Coplin also made cultures from the heart blood and spleen, and obtained a coccus culturally and morphologically identical with the staphylococcus pyogenes aureus. Sections through the brain showed that the lesion consisted of an area containing considerable hemorrhagic extravasation that was surrounded by a layer of polynuclear cells, in the midst of which there were numerous masses of cocci. The surrounding brain tissue showed swelling of the ganglion cells and thickening of the glia. The case, therefore, is a rather interesting example of acute malignant endocarditis, due to staphylococcus infection, and associated with a focal encephalitis. In view of the existence of a chronic osteomyelitis of the right tibia it is perhaps permissible to suspect, in the absence of any other focal lesion, that possibly the organism remained latent in this situation, and then, for some reason, invaded the body, producing the endocarditis and subsequently the various pyæmic manifestations. Unfortunately cultures were not made from the bone lesion.

CASE II.—J. P., white, aged thirty-seven years, was admitted to the hospital on

November 16, 1901, complaining of pain in the back and partial loss of power in the left side of the body. The patient is an intelligent man who had been healthy until a year ago, when he occasionally had attacks which were diagnosed as rheumatism, and a fistula in ano. He has used alcohol to excess; he denies venereal history, and is the father of four healthy children. Three weeks before admission to the hospital the patient, who at that time was in good health, went to bed with a feeling of general malaise and pain in the back and limbs. On the following morning he discovered that he was unable to use the left side of the body; speech was thick; he still had pain in the back and also in the left arm and leg. Physical examination on December 7th showed slight inequality of the pupils, the right being the larger, although both reacted to light and accommodation. The tongue was protruded in a straight line; the supra-orbital reflex was equal on both sides; there seemed to be slight impairment of motion on the left side of the mouth. Speech was defective, the utterance was thick, and there appeared to be special difficulty in pronouncing the letter "R." This, the patient says, has developed since his present sickness. The platysma moved freely on both sides and there was slight rigidity of the muscles of the neck on the left side posteriorly. There was permanent spasticity of the muscles of the right arm, and of all the muscles controlling the left wrist-joint; although the flexors were more involved, and the arm was therefore held completely flexed with the wrist bent at right angles. There was no rigidity of the fingers of the left hand, excepting some spasticity of the long extensors of the thumb. The muscles appeared to be slightly wasted; the grip was weak; the movements of the arm were weak, and the patient was unable to extend it beyond a right angle, although it could readily be extended by the application of force. The biceps jerk was diminished; the triceps jerk was slightly exaggerated, but the muscles did not show mechanical irritability. There were no sensory disturbances in the arm, and all the muscles of the arm and shoulder responded readily to faradic electricity. Aside from the hampering effect of the spasticity, motion was normal, and there was no evidence of inco-ordination. The stereognostic sense was normal. Whenever an effort was made to move the left arm the patient moved the right arm with it in a somewhat similar manner, and this associated movement could not be controlled voluntarily. The right arm was entirely flaccid; movements were normal and were not accompanied by movements of the left hand. The little and ring fingers were persistently flexed on the palm, and there was wasting of the thenar and hypothenar and interosseii muscles. This was evidently due to an injury to the ulnar and median nerves received fifteen years ago, the scar of which was still distinct. The thumb, index and middle fingers were capable of free movement, and the grip was fairly strong. There was slight numbness of sensation of the ulnar side of the hand. The arm reflexes were normal; sensation was entirely normal. The left leg was distinctly spastic; it was held habitually in a position of extreme extension. The knee-jerk was greatly exaggerated; there was persistent patellar clonus, and a rapidly exhausted ankle clonus. Babinski's phenomenon was present to an extreme degree; the plantar reflex was normal. The movements of the leg were executed slowly and clumsily, but without any evidence of inco-ordination. The toes did not resist passive movement in any direction. Sensation was everywhere normal; localization was good, and the muscle sense was not destroyed. When the thigh was flexed upon the abdomen the leg could not be extended beyond an angle of 90°; if the patient sat on the edge of the bed it was impossible to extend the left leg beyond a perpen-

dicular position. Whenever this sign was tested the hamstring muscles, which were soft and relaxed when the leg was extended, became hard and rigid. The spasm was not associated with any pain Kernig's sign had been tried daily since it was first elicited, and the angle through which the leg could be moved diminished from an estimated 115° on November 22d, to between 90° and 95° on December 6th. The right leg was flaccid; the knee-jerk was exaggerated; patellar clonus could not be elicited, and there was no ankle clonus. The Babinski reflex was distinct and characteristic; there was no impairment of co-ordination and no disturbance of sensation. The muscles of both legs responded readily to faradic electricity. The muscles of the ulnar side of the right hand failed to respond. The abdominal reflex was present and apparently normal. The patient states that his memory is not as good as it formerly was; he has occasional emotional attacks apparently due to a realization of the serious nature of his disease, but is otherwise cheerful and exceptionally intelligent. He has complained of diplopia ever since his admission, and an examination by Dr. de Schweinitz on December 4th showed that the vision in each eye was reduced to 5/15, and there was a paresis of 8 prism degrees of the left external rectus. The eye-grounds were normal, and there were no changes in the form fields.

On December 20th, the condition of the patient had improved considerably. The diplopia was rarely present excepting at night; the rigidity of the left arm persisted; both knee jerks were moderately exaggerated, and there was slight spasticity of the left leg. Kernig's sign had completely disappeared, and in spite of the spasticity it was possible to extend the left leg completely. The patient was able to walk with assistance; speech showed distinct improvement, but he was still very emotional.

The nature of this case is somewhat obscure, The lesion is evidently located in the upper portion of the medulla on the right side, and extends far enough forward to involve the nucleus of the left abducens. It cannot be complete because the paralysis is not complete on the left side, and it must be limited because there is no disturbance of sensation. Tumor was at first suspected, and the patient stated that at the commencement of his attack he had headache and vomiting; this, however, soon disappeared, and the absence of choked disks rendered the diagnosis of tumor unlikely. There is no reason to suspect embolism of one of the branches of the basilar artery, but thrombosis of this artery or syphilitic disease of the artery causing partial degeneration in the surrounding tissue cannot be excluded. The sudden onset of the attack leads me to suspect the possibility of a non-suppurative focal encephalitis of the type described particularly by Nonne.

The patient has received potassium iodide in increasing doses since admission. There is, however, no other reason to suspect a syphilitic lesion, for the patient has no recognizable signs of syphilis. The most interesting feature is the presence of Kernig's sign while the symptoms were severe, and its disappearance as improvement progressed.

The nature of the mechanism by which Kernig's sign is produced is still obscure. Kernig himself did not attempt any explanation, but was content to prove that it was not due merely to increased intracerebral pressure, nor to pressure upon the sciatic nerves. Friis suggested that it was due to irritation of the corda equina by the infected

cerebrospinal fluid. Of course, the occurrence of the sign in purely cerebral lesions renders this view untenable. Henoch spóke of it as a reflex manifestation. I do not know exactly what he means by this, but there can be no doubt that the position of the thigh which stretches the flexor muscles evidently produces at a certain point a contraction in them. To call this a reflex does not explain the sign. Exactly the same view has been maintained by Bull, who called attention to the fact that it is difficult fully to extend the leg when the thigh is at right angles to the trunk, and regards the sign merely as an exaggeration of this normal condition. Netter and Herrick, who next to Kernig and Friis have made the most extensive investigations upon this subject, merely discuss the theories that have been suggested by others. Widal and Merklen look upon it as a manifestation of irritation of the spinal meninges, but do not attempt to explain how this irritation produces the very peculiar phenomenon. Chauffard, the most recent author to discuss its nature, explains it as an exaggeration of normal phenomena, due to a hypertonicity of the muscles. It is closely analogous—according to him—to the retraction of the muscles of the neck and back, and he therefore defines it as a contraction mono-regional or multi-regional affecting physiologically predominant groups of muscles, and occurring in attitudes which normally bring these muscles into play.

In the case that I report the common feature was the spastic paresis of one side of the body due to unilateral cerebral lesion. In both cases this spasticity did not produce any retraction of the hamstring muscles when the leg was extended, but gave rise to all the characteristic phenomena of Kernig's sign when the thigh was flexed upon the trunk. In the majority of other cases in which this sign occurred there is reason to suppose a certain degree of irritation of the pyramidal tract associated with depression of its conductile power, that is to say, a condition similar to the one that existed in both of these cases, a spastic paresis. And it seems not unreasonable to suppose that more extensive and careful observations will show that in the majority of cases Kernig's sign may be regarded as one of the symptoms of a partial lesion of the pyramidal tract, that is to say, a lesion which does not destroy it but which prevents its complete functional activity. This does not serve to explain its mechanism, unless we assume with Bull and Henoch that it is merely an exaggeration of normal conditions; but to assert this positively is of course at present unwarranted. Its mechanism is still

as uncertain as is the mechanism of Babinski's sign, which in some respects is analogous to it, but apparently is present when the lesion of the pyramidal tract is much more severe than is compatible with the existence of Kernig's sign. Kernig is the only author who states that the arms may be affected by similar condition. In the two cases which I report there was a flexor spasm in the arms on the affected side, but in both cases this could be overcome by force, was persistent, and bore no definite resemblance to the manifestations in the leg.

It is of course obvious that it is not a lesion of the meninges but of the subjacent nervous substance that causes the occurrence of Kernig's sign. Practically all cases of leptomeningitis are associated with a more or less severe degree of encephalitis. As far as I know, however, the occurrence of the sign in the course of a typical case of encephalitis has not been recorded. In Nonne's series of cases no mention whatever is made of it, although he distinctly states that in one or two cases symptoms of meningitis were not present. Whether this includes the sign or not is of course impossible to determine. It is impossible to say whether the sign is frequently present in encephalitic lesions involving the pyramidal tract or not; that it may occur in such cases, Case I. sufficiently proves, and Case II. supports the affirmative view, although, of course, the diagnosis has not been confirmed by autopsy. At any rate, I think the following conclusions are justified: First, Kernig's sign may occur as a symptom of focal encephalitis, and in this condition may be present upon only the opposite side of the body. Sometimes it is associated with spastic paresis of the leg upon that side. Second, in these cases there may be a persistent tonic spasm of the flexor muscles of the arm, which, however, does not resemble Kernig's sign in its mechanism. Third, the most reasonable explanation of Kernig's sign that we have at present is to ascribe it to an irritative lesion of the pyramidal tract that diminishes but does not destroy its functional activity.

At my request, my resident physician, Dr. Shields, has made careful studies of 100 cases at the Philadelphia Hospital, with reference to the presence of Kernig's sign The results of these studies are appended to this article. In addition, Dr. Clark has, at my request, reported three cases of meningitis, all confirmed by autopsy, in which the sign was absent during the entire period of observation.

References.

Blümm. Münchener medicinische Wochenschrift, 1889, p. 446.
Bull. Berliner klinische Wochenschrift, 1895, p. 772.
Chauffard. La Presse Médicale, April, 1901, p. 153.
Dieulafoy. Clinique méd. de l'Hôtel de Dieu, 1898 and 1899, p. 347.
Friis. Copenhagen Thesis, 1887. Quoted by Netter.
Friis. Ugeskrift for Laeger, 1892, pp. 407 and 431. Quoted by Netter.
Henoch. Charité Annalen, vol. ii., p. 575.
Herrick. The American Journal of the Medical Sciences, 1899, vol. cxviii., p. 35.
Kernig. Berlin. klin. Wochenschrift, 1884, p. 829.
Netter. La Semaine Médicale, 1898, p. 281.
Nonne. Deutsche Zeitschrift für Nervenheilkunde, vol. xiv., p. 1.
Osler. Canadian Journal and Review, 1899, p. 447.
Packard. Archives of Pediatrics, April, 1900, p. 259.
Thyne. Lancet, February, 1901, p. 397.
Widal and Merklen. Bull. et Mém. de la Société Médicale des Hôpitaux, 1899, p. 899.

REPORT OF ONE HUNDRED CASES, ALL NON-MENINGITIC, EXAMINED FOR KERNIG'S SIGN.

By WILLIAM G. SHIELDS, Jr., M.D.,
RESIDENT PHYSICIAN TO THE PHILADELPHIA HOSPITAL.

This report is to show the result of the examination of 100 non-meningitic cases, both febrile and afebrile, for the presence of Kernig's sign. In every case both legs were examined for the sign in both erect and recumbent postures, excepting when the condition of the patient would not admit his sitting up. In 5 cases Kernig's sign was present; 3 showed the sign unilaterally, and 2 bilaterally, 1 case of uræmia and 1 case of typhoid fever, and it is interesting to note that in both of these cases the sign could not be obtained after recovery. Kernig's sign persisted in the 3 remaining cases—2 cases of right-sided hemiplegia, and 1 of typhoid fever—and in all it was obtained on only one side. The case of typhoid fever is still quite ill, and it is possible that the sign will have disappeared by the time the patient has regained her normal condition. In both cases of typhoid fever which showed the sign, delirium was marked and persistent. This predominance of mental symptoms may have been an indication of febrile or toxic cerebral irritability, which might have in some manner been accountable for the presence of the sign.

Number of cases of each.	Diagnosis.	Right leg.	Left leg.	Angle of extension.
1	Cirrhosis of liver	—	—	—
(Case No. 4) 1	Hemiplegia (right)	+	—	115°
(Case No. 5) 1	Hemiplegia (right)	+	—	120
3	Influenza	—	—	—
2	Malaria	—	—	—
8	Pneumonia	—	—	—
4	Rheumatism	—	—	—
48	Senile	—	—	—
25	Tuberculosis	—	—	—
(Case No. 1) 1	Typhoid (B. E. P.)	—	+	105
(Case No. 2) 1	Typhoid (J. W.)	+	+	110
3	Typhoid	—	—	—
(Case No. 3) 1	Uræmia (J. C.)	+	+	110
1	Uræmia	—	—	—
100				

The table shows the diagnoses of the cases and the number of each examined. The condition in each leg with regard to the presence or absence of Kernig's sign is noted, and in those cases in which the angle of extension was 120° or less this fact has been stated. Brief reports of the 5 positive cases are given.

CASE I.—C. E. P., girl, aged thirteen years, was admitted to the hospital with a history of having been ill for about five weeks. She suffered from malaise, headache, pain in the chest, abdomen and legs, and for about a week she had had epistaxis, and nausea and vomiting for about three days. There were constipation, cough, and expectoration of a mucopurulent material. Two other persons were ill with typhoid fever in the same house. The patient was pale, the pupils were large and reacted to light and distance. The tongue was dry and coated; there was marked hyperæsthesia on the cutaneous surface, especially the arms and legs; high temperature, and a roseolar eruption on the chest and abdomen. The spleen was enlarged; Kernig's sign was present on the left side, the angle of extension not exceeding 105°. The leucocyte count was 5600. The Widal reaction was positive. On the third day after admission a loud systolic murmur was heard at the apex, and the pulmonic second sound was accentuated. Two weeks later she developed otitis media, and about the same time suppuration of the axillary glands. The patient gradually improved, and at present is convalescent, but Kernig's sign still persists in the left leg, the greatest angle of extension being 105°. The spasm is not painful.

CASE II.—J. W., white man, aged thirty-six years, an iron-worker by occupation. Patient when first seen was treated for alcoholism. After his acute alcoholic symptoms with consequent gastritis had subsided, he continued to have elevated temperature. His blood was examined for the Widal reaction and a positive report returned. He had only three typical typhoid spots distributed over the abdomen. He had constant pain in the head and limbs. Constipation was present all through the attack. No enlargement of the spleen could be determined. The tongue was thickly coated with a yellowish-brown fur. Delirium was present in a marked degree for a considerable time. The subsequent course of symptoms, signs, and temperature proved it to be an undoubted case of typhoid fever. The patient showed Kernig's sign typically on both the right and left sides during the height of the disease while lying in bed. Later in the course of the disease, when the patient's condition warranted his being placed in the erect posture, the sign was obtained when he sat on the edge of the bed. When lying in bed the greatest angle of extension was 105° on each side, whereas, when he sat up ten days later the legs could be extended to an angle of 115° with the thigh. This man recovered completely from his enteric attack, and at the end of convalescence Kernig's sign was not present on either side.

CASE III.—J. C., aged sixty-one years, a bricklayer. On November 3, 1901, the patient was admitted suffering from uræmia. He was unconscious, he could not be aroused, the pulse was rather small and fairly rapid. The eyes were rotated upward and to the left, the pupils being somewhat contracted. No reaction to light could be ascertained. The skin was dry and hot. The body temperature was somewhat elevated; the tongue was dry, brown, and hard; the urine, obtained by catheter (6 drachms), showed a large amount of albumin, with numerous

hyaline and granular casts. He was treated with ordinary uræmic therapeutics and rapidly regained consciousness. In two weeks' time he had improved so much that he required no special treatment or attention. When first examined he showed Kernig's sign on the right and left sides, the greatest angle of extension being about 110°. On the eighth day of observation this sign could not be elicited on either side. This patient was only examined for the sign when he was lying in bed, the thigh being flexed upon the abdomen at an angle of 90°.

CASES IV. and V.—The two hemiplegics referred to were both cases in which the mentality was below par. They were both rather typical cases of right-sided paralysis, which condition had lasted in one case for over twenty years, and in the other for about fifteen years. The mode of onset as well as the subsequent course of the affection could not be ascertained. They were examined, both when sitting up and in a recumbent posture, and Kernig's sign was found present on the right side in each case. In one case the greatest angle of extension was 115°, and in the other 120°. In both cases when the thigh was not flexed the leg could be extended to an angle of 180°.

In concluding this report of 100 cases it is essential to state that in no positive case was there any joint involvement either osseous or tendinous; also that in all these cases the legs could be fully extended, and when the thigh was flexed the tendons became hard and tense. I have called no case positive in which the angle was more than 120°. The only case on this list in which the presence of Kernig's sign might have proved misleading by resembling meningitis was Case I., in which there were delirium, elevated temperature, rapid pulse, a red eruption over the chest and abdomen, with hyperæsthesia of the arms and legs. A leucocytic count of 5600, with positive Widal reaction, and two persons ill with typhoid fever in her home at the same time, made the diagnosis clear.

THREE CASES OF MENINGITIS IN WHICH KERNIG'S SIGN WAS PERSISTENTLY ABSENT.

By F. S. CLARK, M.D.,

RESIDENT PHYSICIAN, PHILADELPHIA HOSPITAL.

(From the service of Drs. Hughes and Salinger, of the Philadelphia Hospital.)

CASE I.—D. C., Italian laborer, aged twenty-two years, had a severe chill associated with pain in the abdomen and headache. In a short time he became unconscious, and when admitted to the hospital one day after the onset, his eyes were open and staring, the pupils were dilated, the right slightly larger than the left. Both reacted to light and accommodation. There was some rigidity of the muscles of the neck which increased to marked retraction of the head as his symptoms progressed. Photophobia and hyperæsthesia were prominent symptoms. There were herpes labialis, and sordes on the teeth, but no eruption on the skin. The knee-jerks were normal; Kernig's sign could not be elicited in either leg in the recumbent or erect postures. Lumbar puncture was performed on two occasions, and a turbid fluid containing pus cells withdrawn. Microscopical examination of this fluid for bacteria was negative. There was a leucocytosis of 15,000, and a slight albuminuria with a few hyaline casts. The patient died on the fifth day without regaining consciousness. At the autopsy there was venous congestion of the meninges, and there was an exudate upon the under surface of the cerebellum and on the thoracic portion of the cord. The dura mater was adherent to the skull.

CASE II.—A. H., a salesman, aged twenty-three years, was suddenly attacked with vertigo associated with faintness and nausea. This lasted about five minutes. Similar attacks recurred at intervals, and he also complained of nose-bleed. Three weeks later when he was admitted to the hospital it was noted that his vision was impaired. He complained of dizziness; the gait was staggering and hesitating; the eye-grounds were reported to be characteristic of inherited syphilis, showing extensive exudative disseminated retinochoroiditis. Potassium iodide, however, produced no effect. Speech was hesitating; there was occasional vomiting; the knee-jerks were increased, and patellar clonus was present on the right side, and ankle clonus on both sides. Babinski's sign and Kernig's sign were not present. There was no retraction of the head, no loss of consciousness, but speech became more and more impaired, and the patient died in the fourth week of the disease. The autopsy showed the presence of a subacute tuberculous leptomeningitis. The membranes at the base of the brain were very adherent, and were separated with considerable difficulty. A few old calcified tubercles were found in the lung, and the right lobe of the liver showed some old healed tubercles.

CASE III.—T. B., an Englishman, aged forty-eight years, a tailor by occupation. Two days before admission he had become violent and had destroyed the furniture of the house. On admission he was unconscious, delirious, and there was continual twitching of the hands and feet. The thoracic and abdominal organs were

normal. Four days after the onset of the disease he developed marked retraction of the head, but the reflexes were normal. Babinski's sign was not present and Kernig's could not be elicited. Lumbar puncture was performed on two occasions, and the tubercle bacillus was found in the clear fluid withdrawn. There was slight albuminuria and a few casts. The patient died on the seventh day, and at the autopsy miliary tubercles were found in the pia mater, and tuberculosis of the left lung, the liver, the spleen, and the kidneys.

Briefly summarized, there is one case of acute cerebrospinal lepto-meningitis whose bacterial nature was not determined, and two cases of tuberculous meningitis. In all of these cases Kernig's sign was repeatedly tested and was never present, no difficulty being experienced in any instance in extending the leg beyond an angle of 150°. This extension did not cause undue prominence of the hamstring tendons, nor apparently hurt the patient. The cases confirm the opinion that Kernig's sign is especially unreliable in tuberculous meningitis.

THE TIBIALIS ANTICUS PHENOMENON, AND OTHER ASSOCIATED PHENOMENA IN HEMIPLEGIA.

By D. J. McCARTHY, M.D.,
ASSISTANT NEUROLOGIST.

In the *Deutsche Zeitschrift für Nervenheilkunde*, Bd. xx., 1901, Strümpell called attention to a phenomenon of considerable diagnostic value observed in cases of hemiplegia. This phenomenon consists in a contraction of the tibialis anticus muscle when the patient attempts to lift the paralyzed limb from the floor, the limb being flexed to a right-angle position. Not only is it possible to feel the contraction of the muscle but the muscle is also easily seen from a distance, on account of the prominent position of the anterior tibial tendon. This phenomenon, according to Strümpell, is present not only in cerebral hemiplegias, but also in spinal types of hemiplegia from high cervical lesions, and in paraplegias of a spastic type, with increased reflexes and a condition of muscular hypertonia. It is not seen in the flaccid type of paralysis, such as that of tabes. Closely allied to this phenomenon is another described by Strümpell in the same paper, namely, dorsal extension of the toes on the paralyzed side when the patient attempts to raise the leg held in a straight position from the surface of the bed. This phenomenon is closely allied in its mechanism to the Babinski reflex and to the extensor phenomenon of the toes recently noted by Oppenheim, a phenomenon elicited by irritating with some blunt instrument the calf of the leg immediately behind the edge of the tibia. The extensor phenomenon of the toes noted by Strümpell is explained by the theory that in a lesion of the motor tract the patient is able to move muscle groups as a whole when he is not able to produce a movement in an individual muscle.

The subject of reflexes occurring only in pathological conditions, is of such importance that I have examined the most favorable cases of hemiplegia in the wards of this hospital, and also those at my disposal at the Philadelphia Home for Incurables, and at Dr. Spiller's clinics at the Philadelphia Polyclinic, for the presence of this phenomenon. I have examined in all twenty-one cases of hemiplegia. In all of these cases the reflexes were present and increased on the affected side. Most of the cases were of long standing. The tibialis anticus phe-

nomenon was present and marked in all of these cases. The extensor phenomenon of the toes, when the leg was raised from the bed, was present in all of these cases with the exception of two. I could find in neither of these any special reason why this phenomenon was absent, as the Babinski reflex was present in both cases.

In three cases of hysterical hemiplegia the tibialis anticus phenomenon was absent on the affected side. In cases of poliomyelitis affecting the lower extremities and associated with loss of reflexes the tibialis anticus phenomenon was not observed.

I have examined only a few cases for Oppenheim's extensor phenomenon and found it present in cases where the Babinski reflex was marked.

Von Bechterew has described during the last year a phenomenon occurring in pathological conditions, to which he has given the name Lumbo-Femoral Reflex. This occurs in lesions affecting the dorsal cord, and consists of a sudden flexor movement of the thighs when the lower lumbar vertebræ are percussed. This phenomenon was present in a case of tumor of the spinal cord which came under my observation four years ago, and in which the tumor was found at autopsy to be situated in the lower dorsal cord. At that time an investigation of all the cases of dorsal lesions, associated with increased reflex excitability of the lower extremities, in the wards of this hospital, failed to reveal this phenomenon except in one case, and even that was doubtful. I cannot, therefore, believe that this phenomenon is constant in dorsal lesions. I have recently reported to the Philadelphia Neurological Society a phenomenon similar to Bechterew's occurring constantly in normal children, but affecting a different group of muscles from those described by Von Bechterew. If a child is placed in a recumbent posture, lying flat on the face, and the second, third, or fourth lumbar vertebra be percussed, there occurs a simultaneous contraction of both flexor muscle groups of the thighs (semi-tendinosus and semi-membranosus muscles). This phenomenon, as a normal phenomenon, would appear to bear the same relation to the Von Bechterew lumbo-femoral reflex as does the normal plantar reflex (flexion) to the Babinski phenomenon. This normal phenomenon (contraction of the semi-tendinosus and semi-membranosus) does not occur in any other muscle group by percussion of other areas of the spine. In a recent case, however, associated with a condition of increased reflex excitability, percussion over the lower cervical spine resulted in a contraction

of both deltoids and both triceps muscles. This, however, is the only case in which I have been able to elicit the phenomenon.

At the January meeting of the Philadelphia Neurological Society I reported upon some new pelvic reflexes suggested by the above phenomena. I found that if the proximal tendon (origin) of the adductor magnus be percussed, there results a reflex inward movement of the leg. The same result is obtained by percussing the insertion of this muscle above the knee. In other words, a muscle which has a tendinous origin and tendinous insertion will have a reflex for either end. This is especially true for a long muscle, such as the sartorius. A reflex for either end of this muscle may be obtained in individuals in whom this muscle is well developed by striking either tendon. In a case of traumatic lesion of the spinal cord affecting the second lumbar segments, a reflex movement was obtained in the adductor group on one side at the insertion tendon of the adductor magnus, but was not obtained at the origin tendon. It is quite possible, therefore—I might say probable—that each of these reflexes, that is, the origin and insertion reflex of an individual muscle, has a separate reflex arc, and this fact may be of value in localizing lesions in the spinal cord.

It may be interesting to note in this connection that the phenomenon obtained by percussion of the Achilles tendon is associated with a contraction of the soleus muscle alone. Dr. S. Weir Mitchell has already pointed out that the gastrocnemius is not involved in the production of ankle clonus, and it is not surprising, therefore, that it is not involved in the production of the Achilles jerk.

It may be said, as a general rule, in the study of reflexes that when an individual muscle and its individual tendon can be isolated, and its tendon is sufficiently exposed for the purpose of percussion, such percussion will result in a contraction of the muscle. In other words, a tendon reflex will be produced. When a muscle, such as the biceps of the arm, has a tendinous attachment at either end, a reflex may be elicited by percussion of either tendon, and it is probable, because the sensory nerve supply of these tendons is different, that the reflex arc will occupy different levels of the spinal cord.

The isolation and study of these individual muscle reflex arcs by experimentation and study of pathological lesions will contribute to accuracy in the localization of spinal disease.

A CASE OF FEMORAL THROMBOSIS IN CHRONIC DYSENTERY.

By THOMAS STOTESBURY GITHENS, M.D.

The first cases of thrombosis or phlebitis were reported as occurring in puerperal women. Then came the publication of cases following local infection; and, more recently, thrombosis has been recognized as a complication of infectious fevers and of grave anæmic and cachectic states. A review of the literature shows that very few instances following or complicating dysentery have been recorded. For this reason it has been thought that the report of the following case might prove interesting. I wish to thank Dr. Riesman, my chief at the Philadelphia Hospital, for permission to report the case, which occurred in his wards. The report is as follows:

Thomas J., white, male, aged forty years, born in Wales, single, entered the hospital on March 19, 1901, complaining of abdominal pain and diarrhœa. His family history is negative. He had had typhoid fever and acute dysentery in South Africa fifteen years before. Since that time he has had a chronic diarrhœa, with from twelve to fifteen watery stools daily. He also has severe shooting pains in the epigastric region, which come on about three hours after eating and last almost to the time of the next meal.

Physical Examination.—An anæmic and much emaciated man, of fair muscular development. He has fallen in weight from 165 to 116 pounds within the last three months. Face, anxious and worried. Pupils, slightly unequal; left smaller than right; react to light. Tongue, red, bare, and beefy; dry. Heart and lungs, negative. Abdomen, thin and retracted; resistance and tenderness in epigastrium. Liver and spleen, not large. Stomach tympany extends to umbilicus. No tumor palpable.

The patient remained in much the same condition, although with slight improvement, for two months, during which time his stomach was washed out and found not to be enlarged. Chemical examination was also negative, except for the presence of considerable mucus.

May 28th, we find the following note:—At 4 A.M., while lying quietly in bed, he had a sudden stabbing pain on the inside of the lower third of the left thigh, and within five minutes the leg swelled to its present size. Since then he has had excruciating pains all over the leg, especially when it is moved. He is entirely unable to move it himself. The limb is now extremely swollen. The skin is of a deep red-blue color, which disappears on pressure, but immediately returns when the pressure is removed. This is the condition of the limb from Poupart's liga-

ment to the tips of the toes. The tenderness and tenseness are most marked on the calf and on the inside of the thigh. The saphenous vein is hard and corded, and the blood cannot be squeezed out of it. In addition, the leg, from knee to ankle, is diffusely mottled with hemorrhagic spots, from pin-point to small-pea in size. The larger spots are distinctly elevated, and none of them disappear on pressure. The leg is much warmer than its fellow. There is extreme tenderness over all the muscles and over the popliteal, saphenous, and femoral veins. The superficial veins are enlarged and plainly visible. There is no œdema over subcutaneous bones, and no effusion into the knee.

Mensuration shows	*Left.*	*Right.*
Upper thigh	17½ inches.	15½ inches.
Knee	15½ "	13½ "
Calf	13¼ "	10½ "
Above ankle	8½ "	7¼ "
Base of toes	9½ "	8½ "

The pulse is small, rapid, and thready; 140 per minute; the heart-sounds are feeble. There is some cyanosis of chest and neck. Otherwise his condition remains as before.

May 30th.—The limb is still semi-flexed, and cannot be moved without the most excruciating pain. It remains warmer than the other, and the cyanosis has given place to a pinkish redness. The capillaries of the thigh are injected, and form a dense, visible network. The redness disappears on pressure and returns slowly. The entire leg and thigh pit on pressure, and the muscle masses are still tender. The petechial spots have grown larger; some are the size of a dime.

June 3d.—Tested sensation carefully. The entire leg is hyperæsthetic. The measurements taken to-day show it to be rather larger than before.

From this time on improvement was steady. Walking became possible about two weeks later, although the œdema had not entirely disappeared at the end of a month.

The man had all the appearances of being profoundly anæmic, but a blood examination made June 18th showed 4,500,000 red blood corpuscles and corresponding hemoglobin. We can explain this only by assuming that there was a coincident condensation of blood due to the profuse watery discharges—a condition of anhydremia with oligemia, analogous to that seen in cholera.

Microscopic examination of the stools showed them to consist of almost pure pus. There was seen a small spherical diplococcus, which was the only organism found in cultures; and numerous yeast-cells, but no amœbæ. Rectal examination, though extremely painful to the patient, gave no evidence of malignant disease.

Every effort was made to check the chronic diarrhœa, but nothing had more than a temporary effect. The ineffectiveness of treatment and the high degree of emaciation led Dr. Riesman to suspect that there was more than simple ulceration, and to think that it was a case of polypoid colitis (Colitis polyposa of Ziegler). It was thought that to put the colon at rest by establishing an artificial anus might give the man a chance to recover his health, but he declined operation. He left the hospital four months from the time of his admission. The leg at that time became slightly œdematous after the patient had been standing for a while, but it gave him no pain.

Bruen[1] says that phlebitis is extremely common, not only in typhoid fever and cholera, but also in dysentery; but a careful review of the literature of both thrombosis and dysentery shows very few such cases. As far as I have been able to determine, Laveran[2] and Cambay[3] are the only authors that report thrombosis occurring in this disease. Almost none of the works on thrombosis mention dysentery, and very few of those on dysentery mention thrombosis as a complication. We find in Kelsch and Kiener's[4] *Maladies des Pays Chauds* the following passage: "Arterial and venous thrombi are quite rare in dysentery, and are never found in the portal or mesenteric veins. The general slowing of the circulation is probably the cause in these cases." Cambay[3], in his work on *Dysentery in Warm Countries*, reports one case; and Laveran[2], in an article on the subject, cites four cases. Certain other writers on dysentery mention phlebitis, but none report original cases.

The pathogenesis of phlebitis in dysentery is still doubtful, but it is probable that it is due to a local infection from the digestive tract, to which is added a more or less profound anæmia.

A great deal of light has been thrown upon the pathogenesis of thrombosis by animal experimentation,* which has shown: (1) That a noninfected wound in a vein—even when the vessel is cut half across, as in venesection—is not enough to cause obliteration; and Olliver has shown that when thrombosis occurs after venesection, it is not at the point of injury, but higher, indicating that it is due to infection rather than to trauma; (2) that stoppage of the circulation in a vein by aseptic ligature, even if this is left on for twenty-four hours, is also insufficient; (3) that any solid matter in fine powder, when introduced into the veins, causes immediate thrombosis; (4) that many fluids, *e. g.*, defibrinated blood, injected into the veins, will cause it; (5) that living or dead bacteria, or toxins, introduced into a vein, will cause thrombosis; (6) that living bacteria, even in very small number, placed against the outside of the wall of a vein, will produce thrombosis; (7) that in anæmic blood very slight infections, or even stasis, are enough to produce thrombosis. This has been shown by repeatedly bleeding animals in small amounts, until death has ensued from anæmia; although simple anæmia was not enough to cause thrombosis, it occurred much more readily in anæmic blood.

With regard to its seat, venous thrombosis falls into four classes,

* This article was written before the recent works on agglutinins were published.

according to whether it involves the venæ cavæ, the visceral veins, the cerebral and jugular veins, or the veins of the limbs. In the last case, the usual seat is in the leg; and the favorite veins are the femoral, the internal saphenous, and the popliteal. If either of the latter is attacked primarily, the femoral is generally involved by extension. When the arm is involved the veins attacked, in their order of frequency, are, according to Dabeaux[13], the subclavian, the axillary, the brachial, and the radial. The superficial veins of the arm, according to this author, are rarely affected. The arm is the usual seat in the very rare cases occurring in valvular heart diseases, but most of the brachial cases reported have been in tuberculous patients. Pulmonary embolism is very rare. When more than one vein is affected, the second almost always either is involved by continuity or is a symmetrically placed vein on the other side of the body.

The symptoms of thrombosis are: (1) Pain; (2) change in color; (3) change in temperature; (4) immobility; (5) enlargement of superficial veins; (6) œdema; and (7) petechiæ. The only one of these needing explanation is the sharp, sudden pain; and several theories have been suggested. It was first thought to be due to œdema, but it has been shown that it generally precedes the œdema, and that, although it is the first sign to disappear, the œdema is the last. It has also been thought that the sudden distension of all the veins of the limb causes stretching of the nervi vasorum. It is also possible that the sudden increase in the size of the vein may produce pressure upon the nerve that accompanies almost every artery and vein, and is often in the same sheath with it. This view is upheld by the fact that there is no pain or tenderness in the skin, where the nerve may be pushed aside, but only in the muscles, which are closely surrounded by sheaths of fibrous tissue, preventing expansion. Dr. Riesman tells me that Huber has found touch-corpuscles in the walls of veins, and their presence may go far toward explaining this symptom.

The only other symptom deserving mention is a very rare one, viz., an eruption of petechial spots. This occurred in our case, and I find three other cases reported—those of Trousseau and Peter[6], Trousseau[5], and De Brun[7]. It gives rise to no trouble, and must be carefully distinguished from the dusky mottling which precedes and heralds gangrene.

The usual course of a thrombotic attack is that seen in our case;

but in some cases there is pain over the vein several days before any other symptom appears, and then suddenly the sharp pain occurs and the case runs the usual course. In these cases there is probably a distinct period of phlebitis before thrombosis occurs. In a third class of cases there is an entire absence of pain, the first symptom being œdema, or there being no warning of a local condition until a pulmonary embolism causes a careful examination of the veins to be made. In this class of cases it seems probable, for reasons already stated, that instead of a sudden obliteration of the vein there is a slow formation of a thrombus; so that the vessel is gradually closed, and there is time for the establishment of a collateral circulation before it is entirely shut off.

Thrombosis is not infrequently followed by or accompanied with more or less grave conditions, of which the most important are periphlebitis with thrombosis of the accompanying artery, pulmonary embolism, and pain and œdema continuing after the attack. The first of these conditions is serious, because it is very likely to lead to gangrene of the limb. Pulmonary embolism is by far the most important complication on account of its frequency and because it is almost always fatal.

After a vein is occluded by a thrombus, there is a growth of the clot in both directions, as far as the nearest large branch. The proximal end of the clot tapers off into a conical extremity, which is free in the blood stream, and which is easily detached and whirled away. In some cases the entire clot is swept out of the vein, leaving it perfectly clear. This, of course, occurs only in those cases in which there is no phlebitis, and especially in anæmia. In most cases the clot is probably detached because the limb is moved too soon or too freely; and in this connection it is well to refer again to those cases in which there have been no symptoms of thrombosis until pulmonary embolism has suddenly occurred. In these cases, of course, there had never been any restriction in the motion. A number of such cases are reported by De Brun.

The symptoms of pulmonary embolism are sudden pain in the side, painful cough, very intense dyspnœa and cyanosis, a fluttering heart, and generally syncope. The expectoration, if there is any, is very scanty; and either consists of pure blood or is blood-tinged. The treatment is entirely directed to the syncopal attacks; but if a large

artery is plugged, nothing will save life. The inhaling of amyl-nitrite, and hypodermics of nitroglycerin and of morphia, to relieve the pain and distress, are generally recommended. Dr. Riesman advises hypodermics of camphor.

The prognosis of thrombosis may be considered under three heads: (1) Prognosis of the attack itself; (2) liability to recurrence; and (3) prognostic value. The prognosis of the attack, although favorable, must always be extremely guarded on account of the liability to complications and sequelæ. The only important sequelæ are pain and œdema continuing after the attack. Both of these are quite common; and they would probaly seem even more so if more of the cases reported were continued beyond the time of the acute attack. Trousseau[5] dwells especially upon these conditions, and considers that aching in wet weather is rather to be expected after gouty phlebitis. Œdema continues when the principal vein of the limb is permanently obliterated. In many cases the clot is gradually disintegrated and the bloodvessel again becomes permeable; but in others the clot becomes organized into a fibrous cord, in which case œdema may continue ever after, especially after walking.

What is the liability to another attack in a person that has recovered from the first? It is very common for the two legs to be attacked in sequence, and we must not forget this in giving prognosis; but what is meant here is rather the occurrence of other attacks several months, or even years, after the first. Although a number of cases of this sort have been reported, they are by no means common. Recurrence is especially characteristic of gouty phlebitis, and Trousseau[5] reports the following case due to this cause:

A man, aged fifty-six years, subject to rheumatic attacks, had thrombosis of the left internal saphenous vein, but was not confined to bed, and had no œdema except when standing. Two weeks sufficed to cure the condition. One month later he went to bed on account of a similar attack, and remained there for eight days. The next night he had a precordial pang, which woke him. The next morning his pulse was 100, and there was a morbid sound at the base like dry clapping. The next night he had an attack of syncope, in which he died. There was no autopsy.

Although phlebitis is not very uncommon in chlorosis, there is, as a rule, no succession of attacks; but Kidd[8] reports a case of chlorosis in a girl of sixteen who had three relapses about six months apart,

each of which was accompanied by thrombosis. Wolfner[9] reports a case that he ascribes to over-fatness, but the patient is very likely gouty.

Another question is whether the occurrence of thrombosis in the course of any disease influences the prognosis of the latter. This is of especial importance in tuberculosis; and Dodwell[10] has stated that almost all tubercular patients in whom thrombosis occurs die within three weeks; that the limit is thirty-eight days; that most patients die about seventeen days after the occurence of thrombosis; and that in all the cases that have come to autopsy the tubercular process was advancing rapidly at the time of death. He admits that it may occur in the early stages of phthisis, but thinks that then it is largely a coincidence. Hirtz[11] considers that cases in the early stages are not uncommon, and early cases are also reported by Letulle[12] and by Dabeaux.[13] Thrombosis can hardly be said to influence the prognosis of any other disease in which it occurs, except by the fact that it generally occurs in very cachectic or anæmic individuals, and, therefore, betokens a peculiarly depressed condition of the vital functions.

The treatment of thrombosis may be considered, first, as to the treatment of the acute attack; and, secondly, of the complications and sequelæ.

By far the most important point is the fixation of the limb in a state of absolute immobility, as the slightest motion greatly increases the danger of pulmonary embolism. It is best not to elevate the limb, and certainly not to use a pillow, because on a pillow it is absolutely impossible to keep the limb perfectly at rest. The best plan, probably, is to place it between sand-bags for the first week or two, at least. The severe pain may generally be relieved by hot compresses, or by enveloping the limb in cotton batting spread with belladonna ointment. All rubbing or massage of the limb at this time is, however, to be carefully avoided on account of the danger of detaching a piece of clot, and, for the same reason, the greatest care must be taken not to disturb its position while applying any dressing. The pain at times is severe enough to require opium; and in our case two hypodermics of morphia were necessary. The only other internal treatment required is that for the general condition of the patient, such as iodides in syphilis, salicylates in rheumatism, etc.

The after-treatment is often of importance on account of the fre-

quency of continued œdema and stiffness. Massage is probably the best means of relieving these conditions; but it should not be started for at least six weeks after the attack, for reasons already stated. Cheadle[14] recommends electricity for this purpose, and cites several cases in which stiffness and œdema were quickly cured by its use. It may, in some cases, be necessary for the patient to wear an elastic stocking on account of the œdema.

References.

[1] Bruen: Medical and Surgical Reporter, 1885, vol. liii.
[2] Laveran: Arch. de Pharm. et Médicine Militaire, 1884.
[3] Cambay: Dysentery in Warm Countries.
[4] Kelsch and Kiener: Maladies des pays chauds.
[5] Trousseau: Clinical Medicine.
[6] Trousseau and Peter: Clinique Medicale d'Hotel Dieu, 8th ed. Quoted by Schmidt, Paris. Thesis.
[7] De Brun: Paris Thesis, 1883–84.
[8] Kidd: Lancet, 1895, vol. ii.
[9] Wolfner: Berliner klinische Wochenschrift, 1889, vol. xxvi.
[10] Dodwell: American Journal of the Medical Sciences, June, 1893.
[11] Hirtz: Bull. et mem. Soc. des Hôpitaux, June, 1894.
[12] Letulle: Bull. Soc. Clinicale, March, 1878.
[13] Dabeaux: Thesis, Paris, 1882–83.
[14] Cheadle: Lancet, 1898, vol. ii.
[15] Laveran: Boutons de Biskra. Annales de Derm. et Syph., Series 2, vol. i.

REPORT OF A CASE OF PROGRESSIVE ANCHYLOTIC RIGIDITY OF THE SPINE (RHISOMELIC SPONDYLOSIS).

FROM THE NERVOUS WARDS OF PHILADELPHIA HOSPITAL.
SERVICE OF CHARLES K. MILLS, M.D.
BY CHARLES H. HOLMES, M.D., RESIDENT PHYSICIAN.

Many of the cases of this disease which have been reported correspond in all of their symptoms, exclusively, to one or the other type of the affection known, respectively, as the Von Becterew and the Strümpell-Marie types of rhisomelic spondylosis. There are frequently, however, symptoms in any given case which overlap the bounds of one type and infringe upon the domain of the other.

Briefly stated, the Von Becterew type has for its common symptom progressive rigidity of the spine, and for its differentiating features the root pain and the posterior root and posterior cord degeneration. The Strümpell-Marie type has for its common symptom progressive rigidity of the spine, and for its differentiating symptoms the involvement of the large joints, most commonly the hip, next the knee, and rarely the shoulder joints, with more or less marked anchylosis and deformity of the articulatory portions of bones making up such joints. This case, which is now in the Philadelphia Hospital, presents symptoms which are common to both the Von Becterew and the Strümpell-Marie types of the disease.

A. M., aged thirty-seven years, American, a mill-hand, married, the father of two living children. He has lost none, and his wife has had no miscarriages. He was born in Pennsylvania, and lived there all his life. His father died when fifty-nine, his mother at sixty. One brother died from diphtheria when three years old. One sister died in confinement. One sister from unknown causes at twenty. In the family there is no history of tuberculosis, malignancy, or neuropathic condition; no history of rheumatic affection except in the case of his mother, who, from the age of forty years until she died, had such extreme stiffness of the right shoulder joint as to be unable to raise the hand to the head; no other joints were involved, and no painful involvement of this joint. The patient had all the diseases of childhood and an attack of scarlet fever when eleven years old. At the *age of thirty-three* years he had an attack of gonorrhœa which lasted about one

month. Three months later a so-called "chancroid" appeared, which disappeared in three weeks' time without treatment. Has used tobacco and alcohol in moderation, has been a hard worker, but has never been surrounded with the so-called rheumatic tendencies.

Present Illness.—It began when the patient was twenty years old, with shooting pain in the dorsal spine, worse at night, not made worse on effort. Each following day he noticed he could move the head and dorsal spine less freely. These shooting pains lasted three months and have never returned, but the cervical, dorsal, and lumbar stiffness have remained and become progressively worse.

At the age of twenty-four he began to have pain, which was localized to the right hip joint, did not radiate, and was made worse on effort.

At the age of thirty-three the right hip joint began to stiffen, and the pain which had existed previously disappeared. During the same year he contracted gonorrhœa, and in three weeks' time the stiffness of the right hip joint became very marked, and he first noticed pain and stiffness developing in the right knee joint. The pain was dull in character and not attended by any redness or tenderness. Two weeks later the left knee joint became similarly involved. The stiffness in both knee joints has existed ever since that time and become progressively worse. Both legs are in semi-flexion and cannot be extended. After the involvement of both knee and one hip joints he was obliged to sit in a chair continuously in one position, as a direct result of which he thinks pain developed in the left hip joint and both shoulder joints at the same time, and progressively developing stiffness followed in two months' time in all three joints. The final result of this series of events has been the formation of a single piece, as it were, from the head to the knees; semi-flexion of the knee joints with complete inability to extend them; inability to raise the upper arm at more than a right angle to the body; inability to open the mouth more than one inch. Patient states that a slight cough accompanied the onset of the disease, with occasional night-sweats and blood-stained sputum. None of these symptoms exist at present.

Physical Examination.—The patient is a well-nourished man of thirty-seven years; temperature, 98.2°; pulse, 76; respiration, 32. Arteries are slightly atheromatous. Weight, 128 pounds. Height, 5 feet, 5 inches. He is not able to stand or walk without assistance. Body is bent forward to an angle of 45° with the vertical long axis of the skeleton; the thighs are flexed on the trunk to an angle of 30°; the knees are semi-flexed.

The right thigh and knee show slightly more flexion than the left. There is still slight voluntary motion in the right knee joint, but the right hip and the left hip and knee joints are firmly anchylosed. Both ankle and toe joints are free. In attempting to use the legs, as in walking, he moves the extremities in a rotary manner, pushing the feet forward on the floor and twisting the body around. The spine is rigid throughout, but no abnormality in position or shape of the vertebræ can be detected. The patient can sit up only by resting on an inclined surface like a Morris chair. When attempting to turn in bed he pulls himself over with his hands; he cannot turn on the right side, however, (probably because there is more fixation and less power in the left upper arm). Right pupil is of normal size, iridic reflex normal for light and distance. Left pupil obscured by traumatic cataract. Left eye shows slight tendency to external strabismus, with inability for convergence. There is no loss of power in any of the muscles of the face; the left temporo-maxillary articulation shows marked stiffening with limitation of motion. When the mouth

is widely opened the chin is projected toward the left, and the distance between the superior and inferior central incisors is one inch. Movements of the head on the neck are greatly limited in all directions. Head is moved more freely toward the right than in any other direction, but even this excursion cannot be more than three inches from the median line. Patient can be raised on his heels by applying the lifting force to the occiput. The movements of both elbow, wrist, and finger joints are free. The tendon reflexes of the upper extremities are all slightly exaggerated about equally on both sides. The arms cannot be raised to more than a right angle to the long axis of the body in any direction. There is no loss of sensation (pain, tactile, or thermic) in the arms or in any other part of the body. With the exception of slight roughening of the first sound of the heart at the apex, the heart, lungs, liver, and spleen show nothing of interest. The thighs are parallel, and both hip joints completely anchylosed; the slightest abduction is impossible. The distance between the lower ends of the femur is three inches, no deformity of either hip joint is discernible. The right knee joint still retains slight mobility, the left is firmly anchylosed; both knees are in semi-flexion; both joints show a globular deformity, bony in consistency. The left is much the larger, measuring five inches in diameter at its largest point. Along the course of the anterior tibial nerve of the left leg there is marked tenderness on deep pressure and hyperæsthesia on merely touching the skin surface. The cremasteric reflex on the left side is slightly more prompt than on the right. The tendon reflexes of the lower extremities are slightly increased on both sides, about equally so on each side. There is no Babinski reflex or ankle clonus on either side.

Common to the two types of this disease, this case has had slowly progressing spinal rigidity, developing in the dorsal region and extending throughout the length of the spine. Subsequently, there developed marked stiffness with partial anchylosis in the hip, knee, shoulder, and temporo-maxillary joints, in this respect resembling the symptoms of the Strümpell-Marie type, with the exception, perhaps, of the involvement of the smaller joint (temporo-maxillary). Unlike the Strümpell-Marie type, the stiffening of the spine and the further involvement of every joint has been preceded by a distinct period of intense pain, described by the patient himself as "shooting," in the spine, and "dull localized" in the other joints. The pain has in every instance disappeared as soon as the limitation in motion of the joint has become marked. A further sensory phenomenon is noted in the area of hyperæsthesia in left leg, which has existed for eight months' time.

Many writers have referred to a possible causative relation between gonorrhœa and rhisomelic spondylosis. Strangely enough, this patient himself dates all of his present trouble to his previous attack of gonorrhœa, and the history will show that none of the large joints were affected until three months after this specific trouble; at the same

time there never have existed any symptoms of an acute gonorrhœal arthritis. The stiffening of the temporo-maxillary articulation on the left side is a symptom which is not of common occurrence. At one time this was so extreme that the patient could take only liquid diet. The right temporo-maxillary articulation has not yet been affected.

The patient is still in the Philadelphia Hospital, and the future course of the disease will be watched with much interest.

DILATATION AND HYPERTROPHY OF THE HEART WITH OLD AND RECENT VALVULITIS.

By HERMAN B. ALLYN, M.D.

The following case illustrates the difficulties in diagnosis met with in many Blockley cases. These difficulties are partly the result of imperfect knowledge of the patient's antecedent ailments, and are partly due to the fact that disease in these people is not limited to a single organ, but invades a number of organs and tissues.

J. J. S., aged thirty-nine years, a native of Philadelphia, a laborer by occupation, was admitted to the Philadelphia Hospital on June 11, 1902, complaining of dyspnœa and œdema of the feet and legs.

Family History.—The father and one brother died of typhoid fever; the mother is much crippled with rheumatism.

Past Medical History.—The patient has had measles, pertussis and typhoid fever. When a boy he had rheumatism accompanied with severe pecordial pain. The entire illness lasted two years, and since then he has had several attacks. Dyspnœa on slight exertion has persisted since the first attack.

History of Present Illness.—About a week before admission to hospital dyspnœa increased and his feet and ankles began to swell. He grew worse until admission, when the patient was in great distress from orthopnœa. The legs were swollen, and the face puffy and anæmic. The pulse on admission was 98 to 100. His general appearance is that of a well-developed and well-nourished man. The face is swollen and skin of a muddy color. The eyes are normal. The tongue is coated and moist; the breath foul.

On June 20th, the following notes were made: There is a very loud systolic murmur heard all over the chest, loudest toward the second right costal cartilage and transmitted into the carotids. There is also a systolic thrill in the same areas, distinct in the suprasternal notch and in the subclavians and carotids. The apex beat is in the sixth intercostal space one inch to the left of the nipple line. At the apex a loud, coarse, rumbling systolic murmur is heard, and a shorter, fainter diastolic murmur. The same diastolic murmur is heard to the left of the sternum, where it is more distinct than at the apex, and it is also heard to the right of the sternum, but there it is not so distinct; and it can also be distinguished faintly at the ensiform cartilage. There is dulness on percussion in the fifth right interspace. The pulse is slow and somewhat irregular, and receding quickly after striking the finger. On friction of the skin of the forehead, capillary pulse is visible. The right femoral pulse seems larger than the left. A (systolic) murmur can be made out in the abdominal aorta between the ensiform cartilage and the umbilicus.

The chest is well developed, and expansion is good. There is some œdema of

the chest wall. The lungs show increased vocal fremitus, and there are fine râles, crepitant and subcrepitant, over the right lung posteriorly and as far around as the anterior axillary fold. The percussion note is dull and higher pitched in the left interscapular space. A systolic murmur is plainly heard here and down the left side of the spine to the second dorsal vertebra. The same is true of the right side of the spine.

The lower border of the left lobe of the liver extends to one and one-quarter inches above the navel. The right lobe does not seem enlarged, but it extends to the transverse umbilical line. The edge is somewhat thickened and rounded. The spleen was not palpable.

There was no diastolic shock over the body of the heart, no tracheal tug, no difference in size of the pupils. No retraction of interspaces in systole was noted.

The urine, on June 12th, had a specific gravity of 1024, contained a trace of albumin and a few hyaline casts.

The pulse was generally slow, ranging from 40 to 60, and the temperature subnormal. The patient had some difficulty in sleeping, and at times he was faint and had pain in the chest.

The diagnosis in this case was dilatation and hypertrophy of the heart, with stenoris and insufficiency of the aortic orifice due to valvulitis; mitral insufficiency, possibly relative, and interstitial nephritis; adhesive pericarditis was not suspected, and apparently gave no signs of its presence. There were two possibilities to be considered, the existence of aneurism and pericardial effusion. Of aneurism there were none of the ordinary signs present—no tumor causing demonstrable dulness on percussion, and pulsation, or tumor suspected from the presence of pressure signs on nerve trunks, such as difference in the pupils and unilateral sweating; no tracheal tug, no diastolic shock. Yet it was possible that there was an aneurism too small to produce pressure signs, or that it affected the descending portion of the arch and so did not manifest its presence anteriorly. It will be remembered that the physical examination showed dulness in the left interscapular space, and a systolic murmur transmitted down the spine. Of the other possibility, pericardial effusion, there was only one sign, dulness in the fifth right interspace close to the sternum. Moreover, it was further possible that he might have dilated hypertrophy of the heart and valvular disease, associated with aneurism and a small pericardial effusion. To clear up the diagnosis Dr. G. E. Pfahler was requested to make an X-ray examination, which he very kindly did. He reported that he found cardiac hypertrophy, with a possibility of pericardial effusion, and tortuosity of the aorta simulating aneurism.

The patient died on the sixth of July, twenty-five days after ad-

mission. At the autopsy the pathological diagnosis was chronic adhesive pericarditis; dilatation and hypertrophy of the heart; old and recent endocarditis of the aortic and mitral valves; interstitial nephritis.

The heart with a portion of the descending aorta weighed 1090 gm. There was marked general adhesive pericarditis. The left ventricle measured 3 cm. in thickness. The aortic valve was thickened and calcareous. On one of the cusps toward the ventricular side was an irregular, cornucopia-shaped calcareous pocket, within which were some recent vegetations. The mitral valve also was atheromatous. There were no atheromatous deposits in the aorta. Just beyond the aortic valve the aorta when laid open measures 8 cm. in width, and 15 cm. from the valve it measures 5 cm.

The main features of this case were recognized during life. The adhesive pericarditis was not suspected and gave rise to no symptoms; the hypertrophy of the heart was amply accounted for by the demonstrable aortic lesion. The mitral valvular murmur was thought during life to be possibly due to relative insufficiency, but the coarse, rumbling character of the murmur is what one would expect from a sclerosed and insufficient valve. On the other hand, I hesitate to make a diagnosis of mitral insufficiency due to valvulitis when aortic insufficiency with a dilated left ventricle are undoubtedly present. In such cases one is apt to find at autopsy only a relative insufficiency.

What surprised me most at the autopsy was the extensive calcareous deposits on the valves. For while the history indicated that the primary attack occurred in boyhood, the man was only thirty-nine years old, and his arteries were not atheramatous. But we meet with such cases at the Philadelphia Hospital in men of the laboring class, whose lot is often a hard one, and who are frequently of intemperate habits; though as to indulgence in alcohol the history in the present instance is silent. With regard to the recent endocarditis, that is well known to be fairly common as a terminal stage of chronic endocarditis. In our patient it gave rise to very little disturbance, and there is nothing in the history to indicate the date of its onset.

CIRRHOSIS OF THE LIVER WITH ASCITES; OPERATION.

By HERMAN B. ALLYN, M.D.

Arthur Derrickson, white, aged sixty-two years; birth-place, New Jersey; residence, Philadelphia; by occupation a laborer; was admitted to the hospital July 31, 1902, complaining of abdominal distention with pain; dyspnœa.

The family history was unimportant. Personal history: Had the usual diseases of childhood; had also intermittent fever when a young man. Has often felt poorly but was seldom bed-fast. For many years he has had attacks of cramp colic—a sudden, intense pain in the abdomen, which did not radiate to shoulder or down the legs, and was accompanied by retching and prostration. Has been a beer and whisky drinker, and occasionally drank to excess. He used tobacco a great deal. Has had four children, three of whom are living and well; one died of brain fever. Had gonorrhœa about thirty years ago, and a venereal sore with secondary manifestations later. Has had rheumatism for many years, but has not been troubled with it in recent years.

When in the hospital in June and July, 1900, the patient's liver was slightly enlarged, and he had fluid in his belly and in both pleural sacs. His chief complaint was of sharp pain in the left side, increased by breathing. There was no albumin or sugar in the urine, and the specific gravity was 1025–1030. The heart sounds were weak; the second, accentuated. There was no enlargement of the heart and no murmurs. The pulse was 82 to 88. There was slight œdema of legs, especially the right. The heart sounds improved under rest and treatment, and the effusions lessened. The patient was dismissed at his own request July 13, 1900.

About two weeks before his second admission the patient was taken with severe pain in the abdomen, which became almost unendurable. There was nausea and retching but no vomiting. The attack lasted for four or five hours, he became unconscious, and when he regained consciousness the pain had ceased. This attack was followed by very frequent stools of greenish mucus passed with tenesmus, and accompanied with nausea and vomiting, giddiness and great abdominal tenderness, aggravated by any movement and by prostration. Following the seizure the abdominal distention increased and was accompanied with dyspnœa and cardiac pain.

The patient is a well-developed and fairly well-nourished white man, sixty-two years old. The pupils are equal and react freely to light and accommodation. The tongue is red, fissured, dry, and tremulous. The pulse is regular, accelerated, of good force and volume, the vessels atheromatous. The skin is dry. The chest is well formed. Respiration slightly accelerated but not labored. The breath sounds are very loud down to the sixth rib where they become distant. Resonance is impaired from the sixth rib down. Heart-apex visible, palpable and forcible in the fifth interspace m. c. line. No murmurs. Abdomen is very much distended. The umbilicus projects; girth of abdomen, 38 inches. There is movable dulness

in the flanks. Liver and spleen cannot be outlined on account of the abdominal distention. There is an inguinal hernia in the right side.

August 1st. Ascitic fluid has increased in spite of hydrogogue cathartics and diuretics. Paracentesis performed, 98 ounces of fluid withdrawn. It is of a clear, greenish-yellow; specific gravity, 1010; albumin, 5 per cent. The patient was relieved by withdrawal of fluid, but it reaccumulated in a fortnight, and on the 14th 312 ounces of fluid were withdrawn by aspiration. Clear, straw-colored fluid; specific gravity, 1006; reaction faintly alkaline; albumen, 7 per cent. The patient was more comfortable the following day, but by the 17th it had again accumulated. Dr. A. C. Wood was invited to see him, and surgical interference was advised by him. The patient had the risk of operation explained to him, but chose to have it performed. He was transferred to the surgical wards on August 18th. During his stay in the medical wards the temperature was generally between 97° and 98⅗°. The pulse was always accelerated, ranging from 88 to 120, and generally being about 100. The urine was examined on several occasions, but no albumen was found and no sugar. The specific gravity was 1024 and 1029.

Derrickson was operated on by Dr Wood, August 18th, and died ten days later. There was no recurrence of fluid. The wound never showed any signs of healthy granulations. He died apparently of inanition and exhaustion. The course of temperature was afebrile, in fact, subnormal temperature prevailed generally, except on the day of death, when the temperature rose to 99⅘°.

THE TRAINING SCHOOL FOR NURSES.

From July, 1888 to April, 1903.

By MARION E. SMITH,
CHIEF NURSE FROM 1888 TO 1993.

When the lamented founder of the Training School for Nurses died on June 3, 1888, there were seventy-seven (77) nurses on duty. There were two courses of training, a one-year and a two-year course. The former was without pay. In January, 1894, the one-year course was abolished by the Board of Charities at the request of the chief nurse, and the two-year course retained. In January, 1898, the course was lengthened to three years, and this still remains, the change having proved to be very beneficial to the hospital service and to the individual nurse. In 1892 the pupil nurses were withdrawn from the women's insane department, where they had been in more or less varying numbers since 1887. In 1893 Miss Roberta M. West, who had been assistant chief nurse since 1887, resigned to take the superintendency of the Emergency Hospital, Washington, D. C. Her resignation was accepted by the Board with great regret. Miss Lydia A. Whiton, a graduate of the school, was elected to fill the vacancy. In July, 1895, the beautiful Nurses' House on the corner of Thirty-fourth and Pine streets was occupied, and the old quarters in a wing of the hospital given up to the patients. Not only was the general health of the nurses benefitted by the separate building, but the class-rooms, reception-rooms, bed-rooms, and generous supply of bath-rooms have all made the lives of pupils and graduates comfortable and pleasant.

Work all over the hospital has greatly increased during the past fifteen years, and to-day the number of nurses on duty, one hundred and five (105), barely suffice to meet the demands of almost the same number of wards as when little more than half were actually in the hospital, about twenty out of the seventy-seven being in the insane department. The men's surgical head nurse was also the clinic nurse.

Now there is a clinic nurse with two assistants always on duty. The receiving ward was given into the nurse's care in 1901, and now has two day nurses and one night nurse. More is expected of nurses to-day than was ten years ago and competition is strong, but the wide range of disease, the many departments, the number of patients, all go to make this wonderful old city hospital a very mine of treasure for the woman who wishes to take advantage of it. Its opportunities and possibilities for making the ideal nurse are unsurpassed, and, perhaps, this more than any other reason makes it so dear to its graduates.

MEMBERS OF THE MEDICAL BOARD.

WITH ADDRESSES, PLACE AND TIME OF GRADUATION, DATE OF APPOINTMENT TO THE PHILADELPHIA HOSPITAL, AND POSITIONS HELD IN OTHER INSTITUTIONS.

In the main this list represents the order of seniority of the different members of the medical board; but in a few instances it does not, as some of the present members are serving for a second period. Some also have been elected during the year, or even at the same meeting of the governing board, and practically the latter do not differ in seniority.

CHARLES K. MILLS, M.D., 1909 Chestnut Street. Graduate of University of Pennsylvania, 1869. Appointed, 1877. Clinical Professor of Nervous Diseases in the University of Pennsylvania.

ROLAND G. CURTIN, M.D., 22 South Eighteenth Street. Graduate of University of Pennsylvania, 1866. Appointed, 1880. Consulting Physician to the Rush Hospital for Consumptives; Visiting Physician to the Presbyterian Hospital; Ex-President of the American Climatological Society.

W. JOSEPH HEARN, M.D., 1120 Walnut Street. Graduate of Jefferson Medical College, 1867. Appointed, 1882. Clinical Professor of Surgery in the Jefferson Medical College.

LEWIS W. STEINBACH, M.D., 1309 North Broad Street. Graduate of Jefferson Medical College, 1880. Appointed, 1885. Surgeon to the Jewish Hospital; Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic.

JOHN H. MUSSER, M.D., 1927 Chestnut Street. Graduate of University of Pennsylvania, 1877. Appointed, 1885. Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Presbyterian and University Hospitals.

HENRY W. STELWAGON, M.D., 223 South Seventeenth Street. Graduate of University of Pennsylvania, 1875. Appointed, 1887. Clinical Professor of Dermatology in the Jefferson Medical College, and in the Woman's Medical College; Physician to the Skin Department of the Howard Hospital.

FRANCIS X. DERCUM, M.D., 1719 Walnut Street. Graduate of University of Pennsylvania, 1877. Appointed, 1887. Clinical Professor of Neurology in the Jefferson Medical College; Visiting Physician to St. Clement's Hospital for Epileptics; Consulting Neurologist to St. Agnes's and the Jewish Hospitals, and to the State Asylum for Chronic Insane of Pennsylvania.

G. E. DE SCHWEINITZ, A.M., M.D., 1705 Walnut Street. Graduate of University of Pennsylvania, 1881. Appointed, 1887. Professor of Ophthalmology in the University of Pennsylvania; Ophthalmologist to the Orthopedic Hospital and Infirmary for Nervous Diseases; Consulting Ophthalmologist to the Philadelphia Polyclinic and College for Graduates in Medicine.

BARTON COOKE HIRST, M.D., 1821 Spruce Street. Graduate of University of Pennsylvania, 1883. Appointed, 1887. Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Orthopedic and Howard Hospitals.

FREDERICK P. HENRY, M.D., 1635 Locust Street. Graduate of College of Physicians and Surgeons, New York, 1868. Appointed, 1887. Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania.

EDWARD MARTIN, M.D., 415 South Fifteenth Street. Graduate of University of Pennsylvania, 1883. Appointed, 1888; served until 1889; re-elected, 1892. Served as Surgical Registrar from 1885 to 1888. Clinical Professor of Surgery in the University of Pennsylvania; Surgeon to the Howard, St. Agnes's, and Bryn Mawr Hospitals; Director of the Department of Health and Charities of Philadelphia.

EDWARD P. DAVIS, A.M., M.D., 250 South Twenty-first Street. Graduate of Rush Medical College, Chicago, 1882. Appointed, 1888. Professor of Obstetrics in the Jefferson Medical College; Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Visiting Obstetrician to the Jefferson and Polyclinic Hospitals.

WILLIAM E. HUGHES, M.D., 3945 Chestnut Street. Graduate of the University of Pennsylvania, 1880. Appointed, 1889. Professor of Clinical Medicine in the Medico-Chirurgical College; Visiting Physician to the Medico-Chirurgical Hospital; Pathologist to the Presbyterian Hospital.

SOLOMON SOLIS-COHEN, M.D., 1525 Walnut Street. Graduate of Jefferson Medical College, 1883. Appointed, 1889. Senior Assistant Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital, to the Jewish Hospital, and to the Rush Hospital.

ORVILLE HORWITZ, M.D., 1721 Walnut Street. Graduate of Jefferson Medical College, 1883. Appointed, 1889. Clinical Professor of Genito-Urinary Diseases, Jefferson Medical College; Consulting Surgeon to Jefferson Medical College Hospital; Surgeon to the State Hospital for the Insane.

ERNEST LAPLACE, M.D., 1828 South Rittenhouse Square. Graduate of University of Louisiana, 1884. Appointed, 1889. Professor of Clinical Surgery in the Medico-Chirurgical College.

GEORGE MORLEY MARSHALL, M.D., 1819 Spruce Street. Graduate of University of Pennsylvania, 1886. Appointed, 1890. Laryngologist of St. Joseph's Hospital and Chief of its Throat Dispensary.

JULIUS L. SALINGER, M.D. Graduate of Jefferson Medical College, 1886. Appointed, 1892.

JOHN M. FISHER, M.D., 222 South Fifteenth Street. Graduate of Jefferson Medical College, 1884. Appointed, 1894. Assistant Professor of Gynecology, Jefferson Medical College; Assistant Gynecologist and Chief of the Department of Diseases of Women to the Jefferson Hospital; Gynecologist to the Phœnixville Hospital.

RICHARD C. NORRIS, M.D., 500 North Twentieth Street. Graduate of University of Pennsylvania, 1887. Appointed Registrar, 1890; Obstetrical Staff, 1894; Lecturer on Clinical and Operative Obstetrics, University of Pennsylvania; Physician in Charge, Preston Retreat; Gynecologist to Methodist-Episcopal Hospital; Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital for Women and Children.

THOMAS G. ASHTON, M.D., 1814 South Rittenhouse Square. Graduate of Jefferson Medical College, 1888. Appointed, 1894.

CHARLES A. OLIVER, M.D., 1507 Locust Street. Graduate of the University of Pennsylvania, 1876. Appointed, 1894. Attending Surgeon to the Will's Eye Hospital; Ophthalmic Surgeon to the Presbyterian Hospital, and to St. Timothy's Hospital.

J. CHALMERS DA COSTA, M.D., 2045 Walnut Street. Graduate of Jefferson Medical College, 1885. Appointed Registrar, 1890. Appointed on Surgical Staff, 1895. Professor of the Principles of Surgery and of Clinical Surgery, Jefferson Medical College; Surgeon to St. Joseph's Hospital.

AUGUSTUS A. ESHNER, M.D., 224 South Sixteenth Street. Graduate of Jefferson Medical College, 1888. Appointed Registrar, 1891, and Visiting Physician, 1896. Professor of Clinical Medicine in the Philadelphia Polyclinic; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases; Physician to the Hospital for Diseases of the Lungs, at Chestnut Hill.

ALFRED STENGEL, M.D., 1811 Spruce Street. Graduate of the University of Pennsylvania, 1889. Appointed Medical Registrar, 1892, and Visiting Physician, 1896. Physician to the University of Pennsylvania and to the Children's Hospital; Professor of Clinical Medicine in the University of Pennsylvania.

W. FRANK HAEHNLEN, M.D., Ph.D., 1616 Walnut Street. Graduate of the University of Pennsylvania, 1882. Appointed, 1895. Professor of Obstetrics, Medico-Chirurgical College; Obstetrician to Medico-Chirurgical and Maternity Hospitals.

ALFRED C. WOOD, M.D., 128 South Seventeenth Street. Graduate of the University of Pennsylvania, 1888. Appointed, 1895. Assistant Surgeon to the University Hospital; Demonstrator of Surgery in the University of Pennsylvania.

ELIZABETH L. PECK, M.D., 819 North Fortieth Street. Graduate of Woman's Medical College of Pennsylvania, 1885. Appointed, 1895. Visiting Physician, West Philadelphia Hospital for Women; Visiting Obstetrician to the Woman's Hospital.

CHARLES W. BURR, M.D., 1327 Spruce Street. Graduate of the University of Pennsylvania, 1886. Appointed, 1896. Professor of Mental Diseases in the University of Pennsylvania.

W. M. L. COPLIN, M.D., 1629 South Broad Street. Graduate of Jefferson Medical College, 1886. Appointed, 1892. Resigned, 1895. Reappointed, November, 1896. Professor of Pathology in the Jefferson Medical College.

JOHN B. SHOBER, A.M., M.D., 1731 Pine Street. Graduate of the University of Pennsylvania, 1885. Appointed, December, 1896. Gynecologist to the Howard Hospital; Associate Gynecologist to the Gynecean Hospital; Visiting Physician to the Bar Harbor Hospital, Maine.

CHARLES H. FRAZIER, M.D., 133 South Eighteenth Street. Graduate of University of Pennsylvania, 1892. Appointed, 1898. Professor of Clinical Surgery in the University Hospital; Surgeon to the University and Howard Hospitals; Surgeon to the Home for Crippled Children.

L. NAPOLEON BOSTON, M.D., 1531 South Broad Street. Graduate of Medico-Chirurgical College, 1896. Appointed, 1898. Demonstrator in Charge of Clinical Laboratory, Medico-Chirurgical College.

CHARLES L. LEONARD, M.D., 1930 Chestnut Street. Graduate of the University of Pennsylvania, 1889. Appointed, 1899.

H. AUGUSTUS WILSON, M.D., 1611 Spruce Street. Graduate of Jefferson Medical College, 1879. Appointed, January, 1900. Clinical Professor of Orthopedic Surgery, Jefferson Medical College; Emeritus Professor of Orthopedic Surgery, Philadelphia Polyclinic; Consulting Orthopedic Surgeon to the Kensington Hospital for Women; Consulting Orthopedic Surgeon, Philadelphia Lying-in Hospital.

HOWARD F. HANSELL, M.D., 1528 Walnut Street. Graduate of Jefferson Medical College, 1879. Appointed, March, 1900. Clinical Professor of Opthalmology, Jefferson Medical College; Professor of Diseases of the Eye, Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmologist to the Chester County Hospital and to the Frederick Douglass Memorial.

HERMAN B. ALLYN, M.D., 501 South Forty-second Street. Graduate of the University of Pennsylvania, 1885. Appointed Medical Registrar, 1898, and Visiting Physician, June, 1900. Instructor in Physical Diagnosis in the University of Pennsylvania; Clinical Professor of Medicine, Woman's Medical College of Pennsylvania.

DAVID RIESMAN, M.D., 1624 Spruce Street. Graduate of the University of Pennsylvania, 1892. Appointed Visiting Physician, June, 1900. Professor of Clinical Medicine, Philadelphia Polyclinic; Instructor in Clinical Medicine, University of Pennsylvania; Consulting Physician to the Jewish Hospital; Neurologist to the Northern Infirmary.

MILTON B. HARTZELL, M.D., 3644 Chestnut Street. Graduate of Jefferson Medical College, 1877. Appointed, July, 1900. Instructor in Dermatology in the University of Pennsylvania.

E. S. GANS, M.D., 711 North Franklin Street. Graduate of Jefferson Medical College. Appointed, 1900. Lecturer on Dermatology in the Medico-Chirurgical College.

JOSEPH MCFARLAND, M.D., 442 W. Stafford Street, Germantown, Phila. Graduate of the University of Pennsylvania, 1889. Appointed, October, 1900. Professor of Pathology and Bacteriology, Medico-Chirurgical College; Pathologist to Medico-Chirurgical Hospital.

SIMON FLEXNER, M.D., 218 South Fifteenth Street. Graduate of University of Louisville, Ky. Appointed, October, 1900. Professor of Pathology in the University of Pennsylvania.

F. SAVARY PEARCE, M.D., 1409 Locust Street. Graduate of the University of Pennsylvania, 1891. Appointed, January, 1901. Clinical Professor of Nervous and Mental Diseases, Medico-Chirurgical College; Neurologist to the Howard Hospital.

WILLIAM G. SPILLER, M.D., 4409 Pine Street. Graduate of the University of Pennsylvania, 1892. Appointed, January, 1901. Assistant Clinical Professor of Nervous Diseases and Assistant Professor of Neuropathology in the University of Pennsylvania; Clinical Professor in the Woman's Medical College of Pennsylvania; Professor in the Philadelphia Polyclinic; Neurologist to the Philadelphia Hospital.

CHARLES S. POTTS, M.D., 1726 Chestnut Street. Graduate of the University of Pennsylvania, 1885. Appointed, January, 1901. Instructor of Nervous Diseases and Electro-therapeutics in the University of Pennsylvania; Assistant Neurologist to the University Hospital; Consulting Physician to the Hospital for the Insane of Atlantic County, New Jersey.

JOHN W. CROSKEY, M.D., 1831 Chestnut Street. Graduate of the Medico-Chirurgical College, 1889. Appointed, January, 1901.

EDWARD B. GLEASON, M.D., 41 South Nineteenth Street. Graduate of the University of Pennsylvania, 1880. Appointed, January, 1901. Clinical Professor of Otology in the Medico-Chirurgical College; Surgeon in Charge of the Nose, Throat and Ear Department, Northern Dispensary.

CHARLES P. GRAYSON, M.D., 251 South Sixteenth Street. Graduate of Jefferson Medical College, 1880. Appointed, January, 1901. Lecturer on Laryngology, University Hospital.

JAMES P. MANN, M.D., 1234 Spring Garden Street. Graduate of Jefferson Medical College, 1887. Appointed, January, 1901.

GWILYM G. DAVIS, M.D., 225 South Sixteenth Street. Graduate of the University of Pennsylvania, 1879. Appointed, January, 1901.

WILLIAM C. HOLLOPETER, M.D., 1428 North Broad Street. Graduate of the University of Pennsylvania, 1877. Appointed, January, 1901. Professor of Pediatrics, Medico-Chirurgical College; Pediatrician to the Medico-Chirurgical Hospital; Visiting Physician to the Methodist-Episcopal and to St. Joseph's Hospitals.

EDWARD E. GRAHAM, M.D., 1713 Spruce Street. Graduate of Jefferson Medical College, 1887. Appointed, January, 1901. Clinical Professor of Diseases of Children, Jefferson Medical College; Physician to the Franklin Reformatory Home.

J. P. CROZER GRIFFITH, M.D., 123 South Eighteenth Street. Graduate of the University of Pennsylvania, 1881. Appointed, January, 1901. Clinical Professor of Diseases of Children, University of Pennsylvania.

J. MADISON TAYLOR, M.D., 1504 Pine Street. Graduate of the University of Pennsylvania, 1878. Appointed, January, 1901. Late Professor of Diseases of Children in the Philadelphia Polyclinic; Assistant Physician, Children's Hospital.

R. H. NONES, D.D.S., 1708 Chestnut Street. Graduate of Philadelphia Dental College, 1885. Appointed Dental Surgeon, January, 1901.

M. H. CRYER, D.D.S., 504 Crozer Building. Graduate of Philadelphia Dental College, 1876. Appointed Dental Surgeon, January, 1901.

I. NORMAN BROOMELL, D.D.S., 901 Crozer Building. Graduate of Pennsylvania College of Dental Surgery, 1879. Appointed Dental Surgeon, January, 1901. Professor of Dental Anatomy, Histology and Prosthetic Technics, Pennsylvania College of Dental Surgery.

THOMAS C. STELLWAGON, JR., D.D.S., 501 Hale Building. Graduate of Philadelphia Dental College, 1897. Appointed Dental Surgeon, January, 1901.

T. MELLOR TYSON, M.D., 1506 Spruce Street. Appointed, 1903. Visiting Physician to the Rush Hospital for the Treatment of Consumption and Allied Diseases; Assistant Physician to the Hospital of the University of Pennsylvania.

R. C. ROSENBERGER, M.D., 2330 North Thirteenth Street. Graduate of Jefferson Medical College, 1894. Associate in Bacteriology, Jefferson Medical College; Bacteriologist to Jefferson Medical College Hospital; Pathologist to St. Joseph's Hospital.

B. FRANKLIN STAHL, M.D., 1502 Arch Street. Graduate of the University of Pennsylvania, 1887. Appointed Registrar, 1896. Physician to St. Agnes' Hospital.

W. A. NEWMAN DORLAND, M.D., 128 South Seventeenth Street. Graduate of the University of Pennsylvania, 1886. Appointed, January, 1898. Associate in Gynecology, Philadelphia Polyclinic; Assistant Demonstrator of Obstetrics, University of Pennsylvania; Assistant Obstetrician, University Hospital.

JOSEPH SAILER, M.D., 248 South Twenty-first Street. Graduate of the University of Pennsylvania, 1891. Appointed Registrar, July, 1900.

WILLIAM C. PICKETT, M.D., 1508 North Fourth Street. Graduate of Jefferson Medical College, 1895. Appointed Registrar, August, 1900.

R. D. NEWTON, M.D., 1504 Arch Street. Graduate of Medico-Chirurgical College, 1896. Appointed Registrar, October, 1900.

JAMES H. MCKEE, M.D., 1519 Poplar Street. Graduate of the University of Pennsylvania, 1892. Appointed Registrar, 1901.

HENRY A. NEWBOLD, Ph.G., M.D., 3907 Walnut Street. Graduate of the University of Pennsylvania, 1893. Appointed, 1901. Instructor in Pharmacy, University of Pennsylvania; Assistant Physician, Nervous Clinic, University *Hospital*; Assistant Physician, Nervous Clinic, Polyclinic Hospital.

OFFICIAL CHANGES.

By Act of Assembly, approved April 8, 1903, the Department of Charities and Correction, which had control of the Philadelphia Hospital, was abolished, and a new Department of Public Health and Charities was created. On May 7, 1903, Dr. Edward Martin was appointed Director of the Department of Public Health and Charities, and on May 14th Mr. George W. Sunderland was appointed Assistant Director. The effect of the Act of Assembly referred to is the consolidation under one head of the functions before performed by the Bureau of Health and the Department of Charities, and the separation from the latter of the Department of Correction.

Official changes among the officers of the hospital are the following: Mr. Robert H. Smith appointed Superintendent in place of William M. Geary; Dr. M. H. Biggs, Chief Resident Physician, succeeding Dr. D. E. Hughes; Dr. F. C. Johnson, Assistant Chief Resident Physician; Dr. W. W. Hawke, Assistant Physician to the Insane Department; and Dr. Mihran K. Kassabian, Director of the Rœntgen Laboratory.

Miss Margaret F. Donahoe has been appointed Chief Nurse, succeeding Miss Marion E. Smith, who resigned.